Transactions

of the

Thirty-Ninth Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

CHICAGO, ILLINOIS
September 9 to 14, 1934

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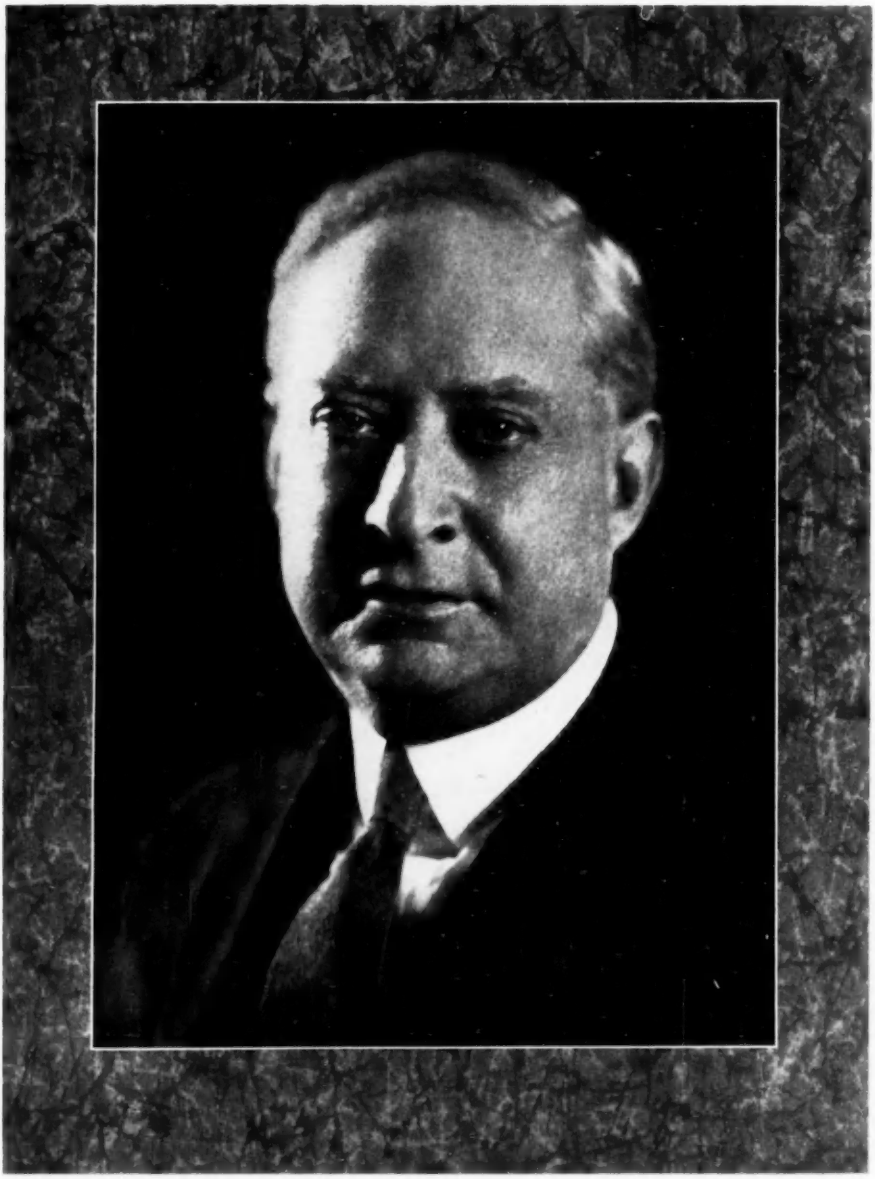


Harris Peyton Mosher

A.B., M.D., Sc.D.

President of the American Academy of Ophthalmology and Otolaryngology, 1929

Dr. H. P. Mosher was born in October, 1867, near Portland, Maine. He is descended from an ancient New England family in the line of Paul Revere. A graduate from Harvard, A.B., 1892, and M.D., 1896, he was surgical house officer at the Massachusetts General Hospital, and, later, house officer at the Boston Lying-In Hospital. During the Spanish-American War, he served as contract surgeon at Montauk Point, Long Island. Thereafter, he studied with Janssen in Berlin and in Schwartze's old clinic in Halle, under Grunert. Three years of general medical and surgical practice in Boston followed.

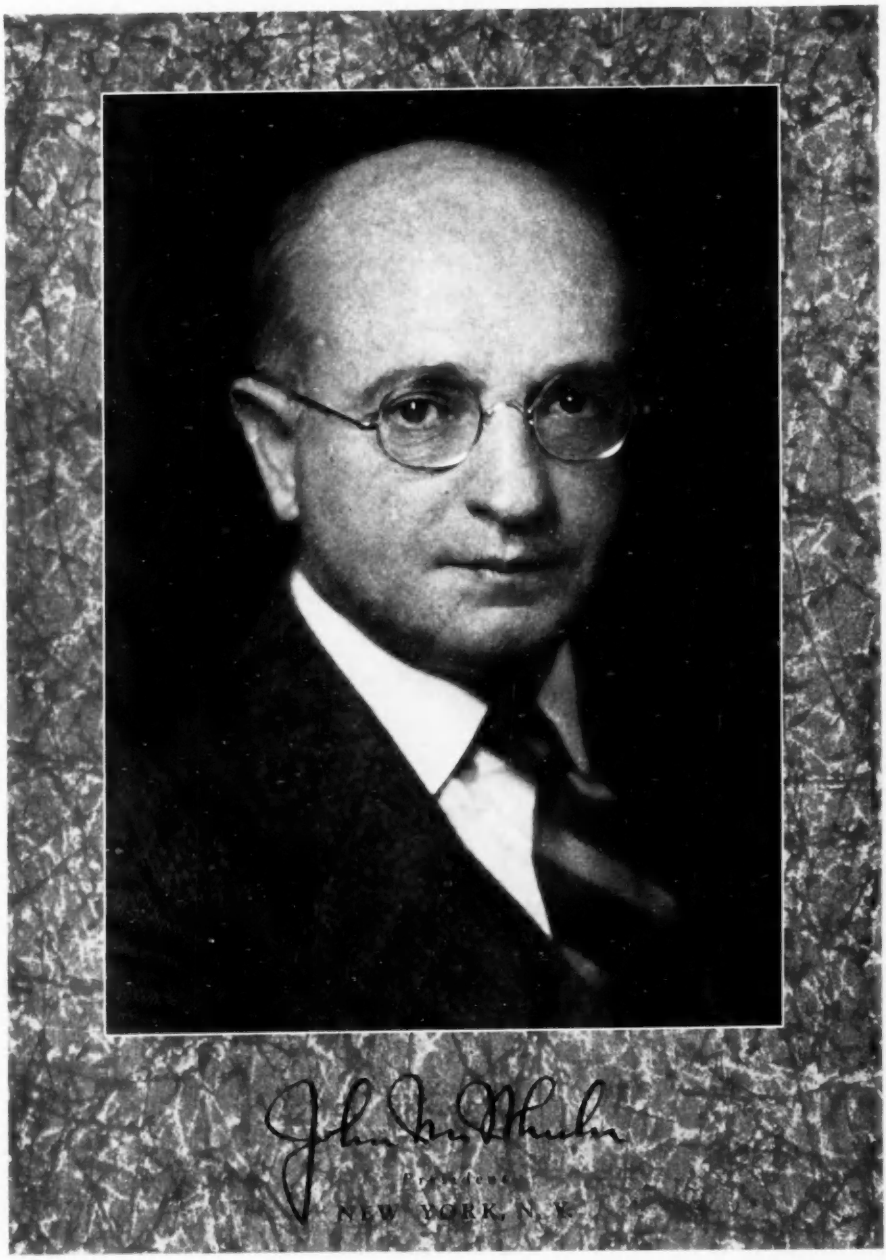
From the start, Dr. Mosher has been a member of the Department of Anatomy in Harvard Medical School. Bringing back Janssen's ideas on sinus anatomy from Berlin, he began a course on the accessory sinuses from wet specimens. This was the beginning of his now famous graduate course which has continued ever since, except for the interruption due to the World War. His skill has made it one of the oldest special courses of this type in this country. Here he originated the method of visualizing difficult structures through drawings and plaster casts. Dr. Mosher's laboratory is recognized as the Mecca for students deeply interested in anatomy and pathology. He considers his best pathological work to have been done on esophageal fibrosis, especially near the lower end.

During the World War, the Surgeon General of the Army detailed Major Mosher, later Colonel, to the assignment of personnel to the otolaryngological services of Army hospitals here and overseas; later in the war, his supervision was confined to this country. He enlisted early and came out late, and his splendid supervisory work afforded the earliest survey of adequacy of training among American otolaryngologists, a preliminary to his presidency of the American Board of Otolaryngology, an office he has held since its foundation. He regards the work of this Board as one of the greatest satisfactions of his career, as it is certainly of paramount importance in graduate instruction in medicine, and has, from its beginning, been part of the Academy program.

Dr. Mosher belongs to all of the national societies in otolaryngology and has served most of them as president; he is a member of the Paris and Vienna Otolaryngological Societies, and corresponding member of the British Laryngological Society. He was the first American chosen to deliver the Semon lecture — in London, 1929 — and then received the Semon Medal; as, also, the American Laryngological Association Medal, 1934. In 1926, the University of Pennsylvania conferred on him the honorary degree of Doctor of Science.

Dr. Mosher's name is attached to the following operations: Intranasal Approach to the Naso-Frontal Duct; Orbital Approach to the Cavernous Sinus; Mosher-Toti Tear Sac Operation; Submaxillary Approach to Deep Cervical Suppuration. He originated the wire gauze drain for brain abscess; paraffin mould for skin grafting the radical mastoid cavity; diagnostic esophageal bag; sight dilatation of the esophagus; and many other procedures and instruments. His publications are numerous, usually illustrated by himself; he has refused, so far, to write a textbook, feeling that his best work lies in teaching. He was the first full-time professor of otolaryngology in this country to devote all his time to the medical school and infirmary.

The Council of this Academy has much pleasure in carrying out the wishes of the membership in dedicating this number of the Transactions to Dr. Harris Peyton Mosher of Harvard University, Boston, Mass.



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Carl Koller, M. D.

Commemorating the Fiftieth Anniversary of His Discovery of Cocaine as a Local Anesthetic

---

Cocaine was first proposed as a local anesthetic by Dr. Carl Koller in 1884. Dr. Koller was then an interne and house surgeon in the Allgemeine Krankenhaus in Vienna.

While experimenting with cocaine as a substitute for morphine in the cure of addicts, he noted the peculiar effect on the tongue when taken orally. A report of this characteristic effect had appeared previously in the literature, but had been lost sight of. Dr. Koller conceived the idea that perhaps cocaine could be used in ophthalmic surgery. Experimentation in Strickler's laboratory confirmed the thought and the first preliminary communication was presented to the German Ophthalmological Society on September 15, 1884. A more elaborate paper was published in the Wiener Medizinische Wochenschrift of October 25, 1884, and was translated in the London Lancet of December 6, and the New York Medical Record.

The Academy honors Dr. Koller upon this fiftieth anniversary of his epoch-making contribution to medical and surgical science.

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Joint Session

The American Academy
of Ophthalmology and Otolaryngology

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Thirty-Ninth Annual Meeting
Chicago . . . 1934

● Seated: Left, PROF. DR. HANS LAUBER, Warsaw, Poland, Guest of Honor; right, DR. JOHN M. WHEELER, New York, President

● Standing: Left, DR. WILLIAM P. WHERRY, Omaha, Executive Secretary-Treasurer; right, DR. WELLS P. EAGLETON, Newark, President-Elect.

ADDRESS OF THE PRESIDENT

BLIND PEOPLE

JOHN M. WHEELER, M.D.

NEW YORK

OVER AND OVER again we have heard, "I'd rather be dead than blind." Sight is the dominating sense, the vital sense, reckoned on a par with life itself. We physicians stand guard over the faculty that people consider the most precious, and we do what is humanly possible to save this faculty for them, or to restore it for them when lost. This might seem to be enough, but we should not allow ourselves to be barred from things beyond this endeavor. There is a place of privilege beyond, that we may rightly enter. I should like to speak of this place, and suggest how we may occupy it to the advantage of others and to our own satisfaction and delight.

Of old, blindness has been associated with feebleness, poverty, ignorance, even filth. The blind beggar has excited sometimes pity, sometimes repulsion. Vergil referred to "A monster, horrid, hideous, huge and blind." Only recently Booth Tarkington said, "A friend, reading to me when I had no vision, incautiously stumbled into a sentence in the work of a new writer of fiction. It was, 'He felt himself as unlike his fellow beings as if he had been blind or an idiot.'"

The blind man was an inferior being, not often chosen for companionship or friendship. As the exception, a few excited admiration. Who can doubt the eminence of Homer, Samson, Milton or Helen Keller! Mark Twain said, "The two most interesting characters of the nineteenth century are Napoleon and Helen Keller. What has she done? She is deaf, dumb and blind. She reads German, French, Latin and Greek. She has passed the most difficult examinations at Radcliffe College."

In the time of Milton, physicians gave particular care to the treatment of the implement that caused the wound rather than to the wound itself. Who were they and their kind to regard

Milton as an inferior being? Who are we to regard Sir Arthur Pearson, Helen Keller, and many other blind and deaf-blind personages of distinction as inferior to us?

How do the blind themselves regard the state that is dreaded beyond all others?

In the Atlantic Monthly of July, 1931, Earnest Elmo Calkins (deaf) and Gordon Lathrop (blind) publish a short debate on which is the greater affliction, blindness or deafness?

Mr. Calkins says he would not give his eyes to have back his ears. Each would have had to experience both blindness and deafness to be an impartial judge. Each, of course, places the highest value on the sense he has left. Most of us who lack a sense wish we had that back, but when it comes to trading another for it, we hesitate. As Hamlet said, "We bear the burdens we have rather than fly to others we know not." Mr. Calkins, without hearing, believes that he is happier, all things considered, than the average man, that he has more resources, that he is never bored. He reads a great deal and says there are enough books in the world to outlast his lifetime. He has taught himself to read French to widen his field of reading. He works with tools and makes all sorts of things, and has learned the complete satisfaction of recreations that do not depend on other people. He says, "I would not go so far as to say that a deaf person is better off than one who can hear, but he *can* be better off than one who can hear. He is certainly better off than one who has lost his eyesight." Blindness has always been the greater tragedy. This is the unconscious but unanimous judgment of humanity. Nature values eyes above ears. She has given us nearly twenty times as many optic nerves as she has aural.

Mr. Lathrop, blind, says that nature is not kind to the blind. Blind animals do not long survive. Blind folk are just as conglomerate a group as would be an equal number of men, women and children picked at random.

Blindness in itself is imprisonment, but blindness to an especially scrappy man or woman constitutes the most thrilling challenge to freedom of the spirit of any affliction visited on man. Blindness may be high adventure and often is. Mr. Lathrop says, "I do not have to be blind in the brain, too." Blindness connotes tragedy. Tragedy brings out the best side of human beings, especially when the tragic figure walks more or less bravely through his part.

Compensation for deafness or blindness does not rush automatically to the afflicted. One must win it. An affliction contains no virtue *per se*. It is a challenge, that is all. Yet this in itself often proves to be a godsend, a spur to the spirit and a gateway to a world more satisfying than the one we knew before.

Blindness in itself is bondage and the loss of physical freedom on account of blindness constitutes doubly-distilled enslavement. Gradually Mr. Lathrop felt the gracious thought of humanity toward the blind and began to like humankind better and humankind responded by reaching out hands on every side. He said, "Then came the dog, Millie, to open my door wide. Millie is to me what Mr. Calkins' electrical device is to him, and more than that. She is companion, friend and servant. She is always available, always efficient when in harness. I am as free as a blind man could be. Millie takes me across the busiest streets, up and down steps, waits for my need of her day and night. Mr. Calkins' ears are in a box. My eyes are in the lovable head of Millie."

"It may be that for every loss there is commensurate compensation. The deaf have not quite so difficult a task as the blind, but the blind have a more thrilling challenge. Potentially, theirs is the greater compensation. This constitutes a potential advantage."

Booth Tarkington says, "It is hard for anyone who has not been blind to realize what a thrill it is not to have to see everything. It was really marvelous not to look at a lot of things and people that you don't want to see."

A few days ago I asked a young lady who had never had light perception whether she would prefer deafness to blindness. "Oh, that's easy," she said, "A blind person can be in on everything, but a deaf person misses so much that is going on around her. She feels out of it, but a blind person can be in on everything."

Eleanor Gertrude Brown, herself blind, says that the average person considers the loss of sight the greatest calamity that can befall an individual, but that she knows of no blind person who would prefer deafness to his own handicap. She says that the late Lord Leicester, who was both blind and deaf in advanced age, found blindness the lesser of the two difficulties. Miss Brown refers to blindness as "inconvenient." She says, "The blind make the adjustment to their handicap perhaps as happily as any group of afflicted people."

Morris Frank, who is totally blind, said to me a short time ago: "Doctor, I don't say this to people, because they would think I was crazy, but I never knew what it was to be absolutely happy until I became blind." For Colonel Frank, loss of eyesight brought a wonderful opportunity for service to others who have lost their sight.

Doctor Villey, a blind scholar, said that he thought with equal intellectual culture there is more equilibrium and judgment with the gifted blind man than with the man who can see.

John Milton was warned by his physicians that he would lose his sight if he continued his exacting work of writing in defense of liberty. He chose blindness, but was profoundly impressed by the sacrifice he made. After he had accepted blindness, he said, "It is not miserable to be blind. He only is miserable who cannot bear his blindness with fortitude, and why should I not bear a calamity to which every man's mind should be disciplined on the contingency of its happening, to bear, with patience, a calamity to the contingency of which every man, by the condition of his nature, is exposed, and which I know to have been the lot of some of the greatest and best of my species? Among those I might reckon many of the wisest of the bards of remote antiquity."

Milton writes, "Shall I not cheerfully bid my eyes keep holiday? Why, in truth, should I not bear gently the deprivation of sight, when I may hope that it is not so much lost as revoked and retracted inwards, for the sharpening rather than the blunting of my mental edge?"

> "So much the rather thou Celestial light
> Shine inward, and the mind through all her powers
> Irradiate, there plant eyes, all mist from thence
> Purge and disperse, that I may see and tell
> Of Things invisible to mortal sight."

That blinded people have heartrending adjustments to make, there can be no doubt. A wonderful opportunity is afforded us physicians to act as guides to our patients through the extremely difficult periods of adjustment, and through the hard days of starting new lives, illumined by "inward light."

I like the graphic way Gordon Lathrop tells of his adjustment and new start in life, in the "Outlook for the Blind," June, 1930. "When I first lost my sight, seven years ago, after thirty-five years of life with good eyesight, I thought that the burden

of responsibility to express friendliness rested on the shoulders of the other fellow. I was suddenly transformed from a successful newspaper man to a penniless blind man with no visible means of support. All I saved had gone in the long fight to preserve my eyesight. When the battle went against me I took the attitude that I had nothing to be friendly about. I had every justification for feeling decidedly sour, I concluded. And I was not going to pretend friendliness toward my fellowman, and the world in general, when I did not feel friendly.

"Consequently, I drove people away from me. I thought that they should know I had strong reasons for feeling glum. I threw the burden on them, not realizing that, if not blindness, every man and woman in the world has had, has now, or will have, some burden of his or her own.

"Humankind loves being friendly, but mightily fears assuming burdens. After a while I learned this. When I learned it, my procedure became one of complete reversal. I became the aggressor in friendliness. It was not my nature to be obnoxiously or effusively aggressive. I had always been rather retiring. But I assumed a state of mind which made for invitation to friendliness by virtue first of seeming friendly, and it worked."

How can the physician be of help to blind people? He may well dwell on this matter. I hope he will discover that in thinking of the problems of the blind and in learning to live a new life with the blind he has found an important means of self-development and that he has enriched his own life in good measure.

Perhaps one of the first things a blind man should learn is that he can play. The knowledge that he can enjoy recreation turns his face away from gloom. He can indulge in rowing, swimming, dancing, gymnastics and many other vigorous recreative activities. He can play cards, chess, checkers and other indoor games to his liking. He can enjoy the radio and the movies. He may like the theater. Even the deaf-blind may be fond of the theater. Madame Maeterlinck says of Helen Keller, "She likes the theater. The vibrations strike her, the waves of sound caress her, the mingled perfumes envelop her, she breathes the hot vaporous air. The heavily charged atmosphere peculiar to a playhouse excites and stimulates her. The unknown agencies that inform her pass to and fro between her and the crowd, filling voids and satisfying her devouring curiosity."

Another thing for the blind man to know is that he can work, and that he can engage in profitable work to his liking. The blind can go in for farming, carpentering, gardening, shoemaking, shorthand, typing, telephone operating, massage and many other things. He stands a good chance at salesmanship. He can keep his own accounts. He can hold positions of responsibility. But, best of all, he can be of help to others who are without sight. He can sometimes be of more help than a sighted person in the problems of adjustment and in actual teaching, for the blind man feels that a fellow blind man understands him and that the fellow blind man knows what he is talking about. It is hardly conceivable that Sir Arthur Pearson could have done his magnificent work with the war blinded as effectively if he himself had not been blind. Of this work he said, "In the days when I could see, I had the direction of some big enterprises, but St. Dunstan's became the biggest individual business that I have ever conducted."

It may be well sometimes for the physician to refer a blind patient to an institution for the blind, but it should not be for a long stay. He may learn something of the art of being blind at an institution where he associates with others without sight, but he should go forward to associate with sighted people. And what a satisfying and instructive thing it may be for the physician to go along with the blind patient in his development!

Probably few blinded persons have failed to wonder if their loss of sight has not been visited on them as punishment for some wrong doing. The mental distress brought on by such meditation may cause torturing self-reproach. A friendly, wise and tactful physician may be able to render real service to a patient who has lost his sight, by listening sympathetically to such an idea brought out in conversation, and by assuring him that he is in no way to blame for his condition, which may come to any of us.

The physician can sometimes do a blind person a great favor by seeing to it that he is not treated like a child or an incompetent, and to see that he is not made an object of pity or undue sympathy. Nor should he ever be treated as an inferior because of blindness. He should be treated as a "regular fellow," and usually he will respond as such. Nothing should be said or done to remind him of his misfortune. And he should know that there is no lack of confidence in him because of his handicap.

Loss of sight in reality may result in an important developmental advantage. There can be no doubt of its effectiveness in sharpening the senses of hearing, smell and touch, and also in increasing general sensitivity. In conversation the voice carries much information to the man without sight. In walking, echoes and street noises carry valuable information. In the house the drip of water, the sound in water pipes, the ticking clock, the creaks in floors, the breath sounds of individuals, and many other sounds usually unnoticed give valuable information for orientation. The sense of smell brings messages, too. The blind person may check up on his whereabouts by the indisputable smell of a bakery, a drug store, a printing office, a shoe shop. He may gain enjoyment in learning to recognize many fragrances never noticed while sight was there. His general sensitivity may develop so that little elevations and depressions will tell his feet where he is, and the skin of his face becomes so sensitive to air currents and changes of temperature that he will have through the skin surface much of value and interest. He may know of the presence of a silent individual or he may be conscious of a wall or other silent obstacle by a canny, undefinable sense. Development of the sense of touch brings a wealth of knowledge and enjoyment to the sightless. He learns to read, write and to divine in wonderful degree through touch. Mrs. Macy says of Helen Keller, "She sometimes wonders if the hand is not more sensitive to the beauties of sculpture than the eye," and in "The Story of My Life," Miss Keller says, "I should think the wonderful rhythmical flow of lines and curves could be more subtly felt than seen."

The blind man should be allowed to do everything possible for himself, and he should not be waited on in any unnecessary particular. With physical and mental activity and with practice and training he will become self-reliant and skillful beyond ordinary belief. He must rely on his memory. He uses it and it increases in capacity. He is prone to engage in thought processes, and these he may perfect. His mental pictures become more important and more meaningful in the absence of his outward vision, and the exercise of mental processes may result in important mental achievement. In fact, blindness sometimes leads one to draw from the great bank of potential mental possibility riches not dreamed of in sight.

The last seven years of the life of the famous Parisian ophthalmologist, Javal, were spent in blindness from glaucoma.

While in darkness, Javal's chief concern was that of rendering assistance to those who had lost their sight. He made many valuable suggestions for physicians who are charged with the care of the blind and those who are losing their sight. For example, he said to his fellow oculists, "Do not, through false ideas of humanity, encourage hope of possible recovery where there is no hope — as by the use of strychnine, electricity, etc. — but, rather, urge the patient to use the little sight which still remains to him in preparing for the total loss which is to come." There is an interesting summary of Javal's helpful suggestions in the 1903 volume of The Ophthalmic Review.

We have heard many times, "If I could only live my life over again." The unsighted person has occasion to learn to live again and to live a new life of surprising wisdom, happiness and service.

Make friends with blind people as with the sighted. How splendid it would be if all of us physicians had lasting, man-to-man friendships, with our blind patients, our blind neighbors!

ADDRESS OF THE GUEST OF HONOR

DIAGNOSTIC AND PROGNOSTIC IMPORTANCE OF OPHTHALMOSCOPY IN RED-FREE LIGHT

HANS LAUBER, M.D.*

WARSAW, POLAND

FULLY APPRECIATING the honour of being invited to lecture at the annual meeting of this distinguished Academy, I have chosen a subject which is surely familiar to the oculists present, but might be less so to the members of the meeting who do not use the ophthalmoscope very frequently. The presence of physicians whose daily round does not include ophthalmoscopic work will justify my offering some preliminary explanations.

When we examine the fundus oculi, we wish first to get objective information on the condition of the retina and the optic nerve, and, secondly, of the choroid and the optic media. The usual ophthalmoscopy in mixed light (electric bulb, gas, kerosene lamp) does not quite fulfil our expectations. Examination of the fundus in such light allows us to see very little of the retina, the vessels, some reflexes, and, sometimes, a suggestion of nerve fibers in the vicinity of the disc. We see the pigment epithelium of the retina, some details of the choroid, and the disc. Study of these elements of the fundus gives us much valuable information and has afforded us the possibility of perceiving many lesions of the inner membranes of the eye. By analyzing the ocular manifestations of extraocular and especially intracranial affections, it supports or even sometimes founds the diagnosis of important and dangerous conditions.

By saying that, in the first place, we try to get information concerning the condition of the retina and the optic nerve, I wish to point out that these two parts of the eye are the principal ones from the standpoint of function and from the anatomical standpoint. This is because they are the directly visible parts of the central nervous system. The retina, being almost per-

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fectly transparent, reflects but very little light, which is practically overwhelmed by the great quantity of light reflected from the pigment epithelium and the choroid. This is the reason why we are not able to see the retina itself under usual conditions.

As the pigment epithelium of the retina and the blood in the choriocapillary membrane are red, they can be seen only by light containing red rays. If we make use of light from which, by means of filters, all the red rays have been eliminated, the red elements must appear black. We avail ourselves of this principle by doing ophthalmoscopy in red-free light. Having explained the principle of the method, I do not believe it necessary to give technical details, and will proceed to describe the phenomena we can see in the fundus by means of this method.

Ophthalmoscopy in red-free light is *the* method of examining the retina, whereas ophthalmoscopy in mixed light shows, mainly, the pigment epithelium and the choroid. Light deprived of red rays is bluish-green in colour. This is also the colour of the fundus seen by the red-free light. The background of the entire picture is formed by the pigment epithelium and the choroid, which appear black. On this background lies the bluish-green retina, which sets off finely the black vessels. In a green retina, the nerve fibers are distinctly visible, so that we can ascertain their presence by direct inspection. We can follow their disposition in the retina and make sure whether they are present or absent, rarefied or œdematous. Further, we can plainly see the yellow colour of the macula, note its changes or its absence. Only in the eyes of individuals of advanced years, the yellow colouring of the lens gives a yellow hue to the entire retina and makes it difficult or even impossible to differentiate the macula from other parts of the retina. The visibility of the yellow colour of the macula allows us to locate the macula quite exactly and to study its relations to pathological foci and also its appearance as well as its reflexes. In consequence of the dark background of the fundus, the retinal reflexes appear much sharper than in mixed light. Study of the retinal reflexes enables us to form a very clear idea of the surface of the retina; for instance, to ascertain the configuration of the macula and the fovea centralis.

The contrast between blood contained in the retinal vessels or extravasated blood appearing black against the bluish-green background is much greater than the contrast between blood ap-

pearing red on a reddish-orange or brownish background, as seen in mixed light. Therefore, minute details of vascular order, changes in the vessels, and partly, also, in blood-circulation are set forth exceedingly well in red-free light. We can easily study changes of the vascular walls and perceive tiny hæmorrhages in the optic nerve or the retina, which would remain perfectly invisible in usual ophthalmoscopy.

These are, in short, the main advantages of ophthalmoscopy in red-free light. This method of examination, without which we cannot do nowadays in serious eye work, however important it may be, cannot be applied by itself and must always be used together with ophthalmoscopy in mixed light. By means of both methods jointly we can obtain a more exact idea of the condition of the internal membranes of the eye, than by either of them applied alone.

Visibility of nerve fibers of the retina gives us the means of forming a correct idea of their condition. Diagnosis of optic nerve atrophy, based on pallor of the disc and thinning of the vessels, frequently is simple, yet it may prove fallacious in certain cases. Cases of extreme pallor of the disc with good vision are known to every experienced oculist. They are quite frequent as a consequence of acute retrobulbar neuritis. We follow the cases and see that the function of the eye, seriously impaired at the onset of the disease, improves rapidly and at the same time the disc grows paler. The following case is an excellent illustration of this phenomenon.

Case I

Defect of the Papillomacular Fascicle after Retrobulbar Neuritis

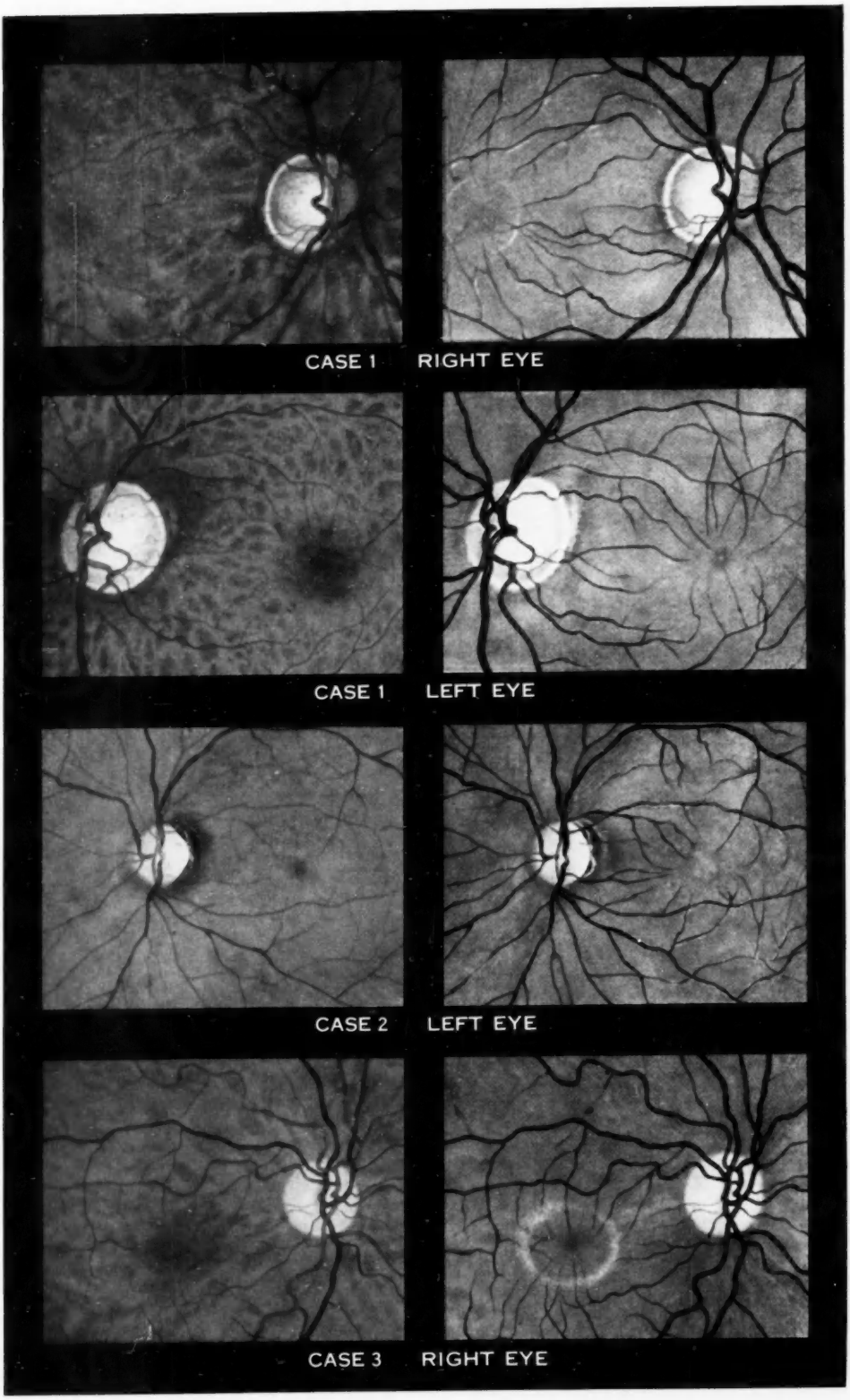
The patient, O. K., aged 24, came, 10 weeks before the pictures were painted, to the clinic of Prof. Meller, because of bilateral, suddenly-developed, almost total blindness. The temporal halves of the discs were pale, though the affection had lasted only a few days. It was an acute retrobulbar neuritis which healed within 6 weeks. The patient was dismissed in the same condition as when his pictures were painted. He states, with absolute sureness, that he saw far better before his disease. Dr. Urbanek showed the patient at the Ophthalmological Society of Vienna on December 12, 1927.*

Right eye: externally normal. In mixed light the disc appears white, only the nasal margin is a little pink. The

* Zeitschr. f. Augenheilkunde, Vol. 64, p. 158, 1928.

dots of the cribiform plate are distinctly visible. In the macular region the small vessels ramify it richly and the minute branches reach almost to the foveola. The fundus shows slight tabulation. The yellow reflex of the macula is of normal size and intensity; the ringshaped reflex is very distinct. In red-free light the small vessels are seen to transgress the reflex and reach the yellow area. The reflexes alongside the vessels are very lively. The nerve fiber striation lacks totally in the region of the papillomacular fascicle. The striation reaches as far as the temporal superior artery and then curves beyond the macula. Below the macula, the nerve fibers extend almost to the inferior temporal vein. Here, too, the nerve fibers take an arcuate course towards a region beyond the macula. Left eye: The disc is also white and shows the dots of the lamina cribrosa distinctly. Examination in the red-free light shows a rarefication of the papillomacular fascicle. Notwithstanding this fact, no scotoma can be found. The yellow area is of normal size; the ring-shaped reflex is absent, as are reflexes along the large vessels. Terminal branches of the vessels invade the macula and fine branches run into the yellow area till they get close to the foveola. This proves that vascularization of the macula does not exclude normal function. Both eyes: V = 10/10. Dark adaptation is reduced to 16/100. With the scotometer of Elliot, with stimuli of 2/1200, a scotoma for red and green could be found between the blind spot and the point of fixation in the right eye. Scotoma in the left eye was not found.

This case is of especial interest, because it proves that "normal vision" amounting to 10/10 can exist simultaneously with a marked defect of the papillomacular fascicle. The white colour of the disc, the disappearance of the nerve fibers of the papillomacular fascicle in the right eye and their strong rarefication in the left eye prove a pronounced lesion of the nerve fibers. The patient has suffered a loss of function which we can't express in numbers, because the acuity of vision before the onset of the retrobulbar neuritis is not known. The present "normal vision" corresponding to the normal average is, however, not normal for the patient. The case proves that good function can be present with marked fundus changes. It is probable that the patient has suffered of retrobulbar neuritis before, but had not noticed the affection, because it did not occur simultaneously in both eyes. Only with the help of this assumption can we explain the pallor of the disc seen at the first examination. The case is also an example of the ingression of ophthalmoscopically visible vessels into the macular area.



LAUBER

(OPPOSITE P. 30)

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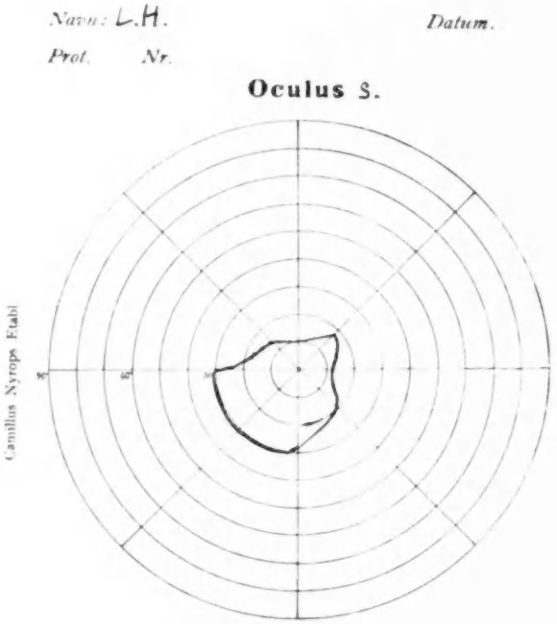
In cases of optic nerve atrophy of different origin, not all fascicles of the optic nerve are affected in the same way. This finds its expression in the irregularities of the field of vision. Examination in red-free light gives us the anatomical substrate of the functional defects, by demonstrating areas in which nerve fibers are absent. The following cases are examples of such conditions:



CASE II

Tabetic Optic Nerve Atrophy

L. H., aged 48. Luetic affection 22 years ago. Mercurial treatment. Vision failing for six months.

Both eyes: Argyll-Robertson pupil, disc quite white with nasal margin a little blurred, arteries thin; fundus otherwise normal. In red-free light the changes in the arteries are more prominent. Some of them are accompanied far into the periphery by white lines and the blood column is only indistinctly visible through the strongly reflecting vascular wall; their diameter is uniformly reduced. The yellow colour of the macular area is normally saturated, though the macula is somewhat larger than usual. Striation

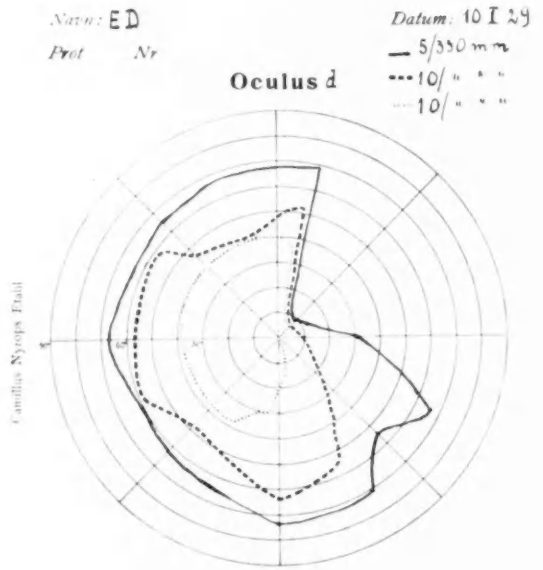
of the nerve fibers is only visible along the superior large vessels. V. = fingers at 3 meters. The peripheral field limits for 10/333 are constricted to between 10° and 25° and the constriction is most pronounced below. For 2/1200ths, the limitation shows a horizontal upper margin only 3° distant from the point of fixation. Neurological diagnosis: tabes in the preataxic stage. Serum Wasserman negative; liquor: Pandy + + +, Nonne + + +, cells 304, Wasserman +. Treatment by infection with malaria did not prevent further progress of the affection.

In this case the examination in red-free light shows the disappearance of the nerve fibers and indicates the possibility of the correlation of their condition with the condition of the field. The fibers are still preserved on the upper temporal side, and on the Bjerrum screen the function can be shown to be still present below and nasally, but completely absent above.

Case III

Temporal Hemiopia by Hypophyseal Tumor

The patient, E. D., aged 34, had had defective vision of the left eye since her childhood and had been operated on for squint at the age of 7 years. In the spring of 1925, the sight of the left eye suddenly decreased considerably. The diagnosis made at the time was most probably retrobulbar neuritis. Opening of the posterior ethmoidal sinuses had no effect and the eye became almost totally blind. In the beginning of October, 1926, visual trouble of the right eye appeared — the patient experienced difficulty in reading. V. was 6/6 and Nieden No. 1 could be read, however, not fluently. There was a small temporal hemiopic paracentral scotoma for colours and a small indentation of the peripheral limit of the field in the temporal superior quadrant. The fundus of the right eye was normal. In the left eye, the disc was absolutely white. The x-ray photograph showed a deepening of the sella, the anterior clinoid processes thin and sharpened. The dorsum sellæ pushed backwards, strongly eroded and only indistinctly visible. Notwithstanding x-ray treatment, complete temporal hemiopia had developed by the beginning of December, 1926. On December 11, 1926, a trephining was performed over the left temporal region in order to reach the tumor from the side. Because of unusual engorgement of the veins, the operation could not be completed and the base of the cranium was not reached. X-ray treatment was continued; followed later by radium treatment from the opened sphenoidal cavities. In March, 1927, the nerve fibers nasal from the disc were less distinctly visible than previously. In August, 1927,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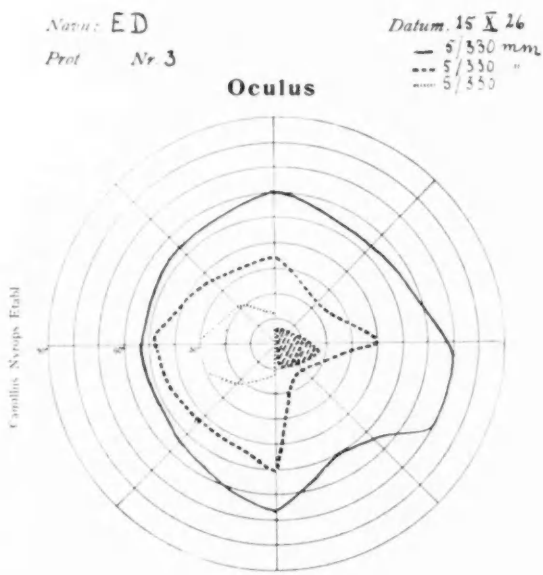
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they had disappeared on the nasal side of the disc. Since March, 1927, there existed an incomplete temporal hemiopia. The upper nasal limit of the field was inclined by 15° to the vertical, the nasal inferior one by 30°. In January, 1929, the field assumed its present condition. The vision is 6/6.

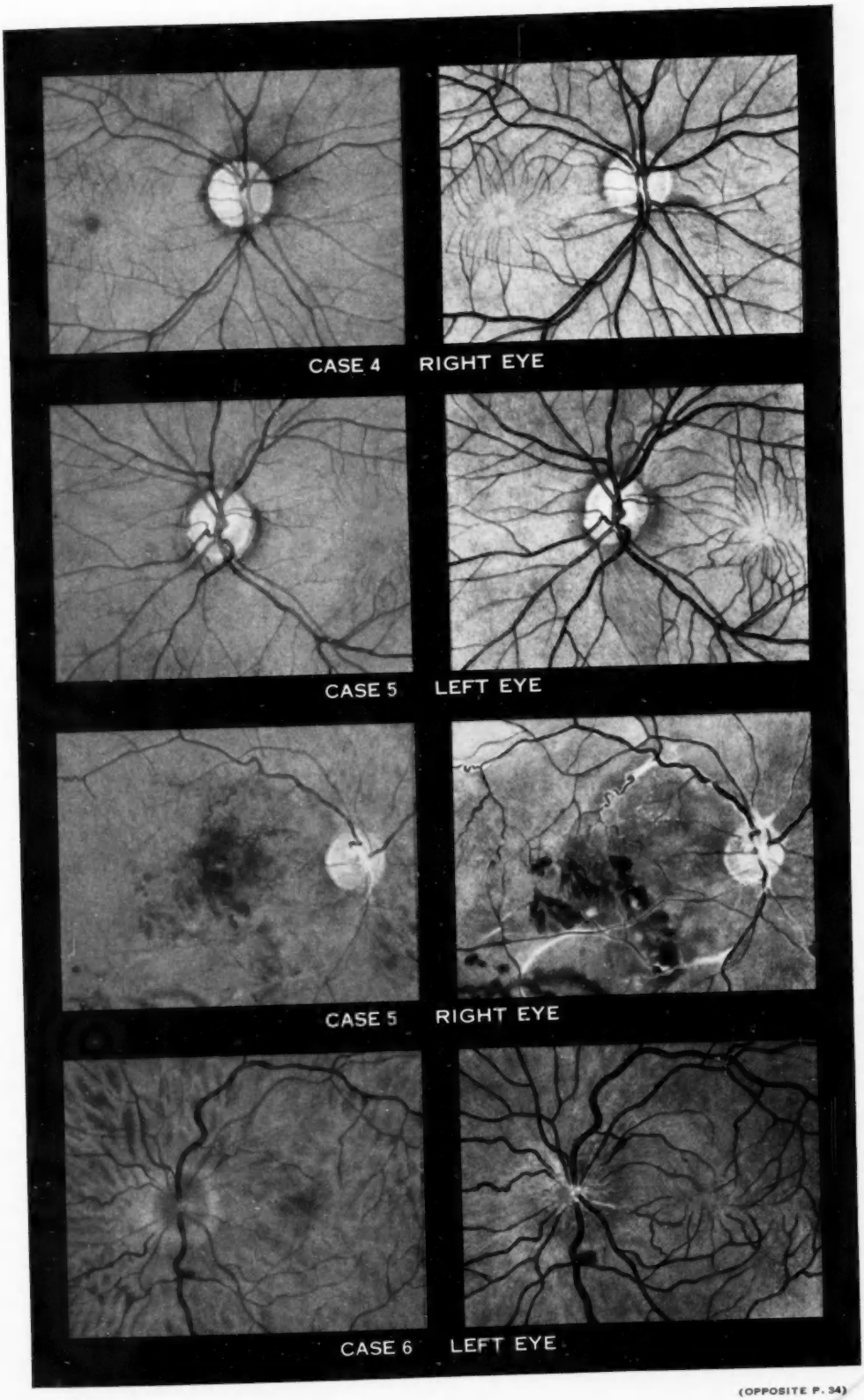
The ophthalmoscope reveals a white disc with sharp limits. The nasal half is quite as pale as the temporal side, which contains a physiological cup with steep edge. The vessels are tortuous, otherwise, normal. The macula is darkly pigmented, the foveal reflex is distinct, the ring reflex has a larger diameter than usual, is broad, but feeble. In red-free light the disc and the vessels show no peculiarities. The macula appears of a brownish colour, the colour being especially saturated in its center. The reflexes correspond to the appearance in mixed light. The nerve fiber striation is distinct on the temporal, upper, and lower border of the disc. In the area of the papillomacular fascicle it is very delicate. It is here absent in a small area immediately above the horizon and in another area a little lower. At the upper and lower margins of the disc it is especially distinct. It seems to end a little to the nasal side of main vessels; on close inspection a very delicate striation is perceptible, which ends a little above the horizon. At the lower margin of the disc the nerve fibers cease sharply in a line inclined 15° to the vertical. From this line to the horizon, no nerve fibers are visible.

The defects of nerve fibers visible in red-free light correspond to the condition of the field. The absence of fibers in the nasal inferior quadrant of the retina corresponds to the defect of the superior temporal quadrant of the field. Mark the symmetry of the limits of the nerve fibers and the field defects. Corresponding to the colour defect in the main part of the temporal inferior quadrant of the field, the nerve fiber striation is very delicate on the nasal superior side of the disc. Here the limit of the colour field corresponds to the limit of the distinct nerve fiber oblique striation. The two small defects of the fibers at the temporal margin of the disc correspond to the crossed nerve fibers going to the macula.

Case IV

Left Homonymous Tractus Hemiopia

K. S., aged 41, 1907 luetic infection, mercurial injections. 1910, iritis. 1915, mercurial injections because of the skin symptoms. In 1923, there appeared in the left



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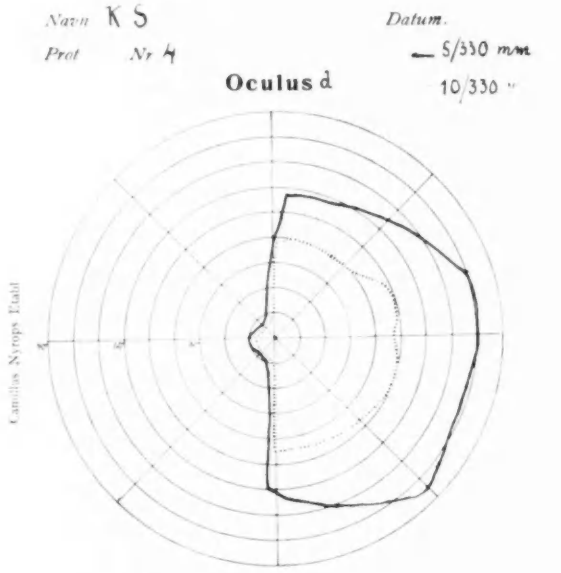
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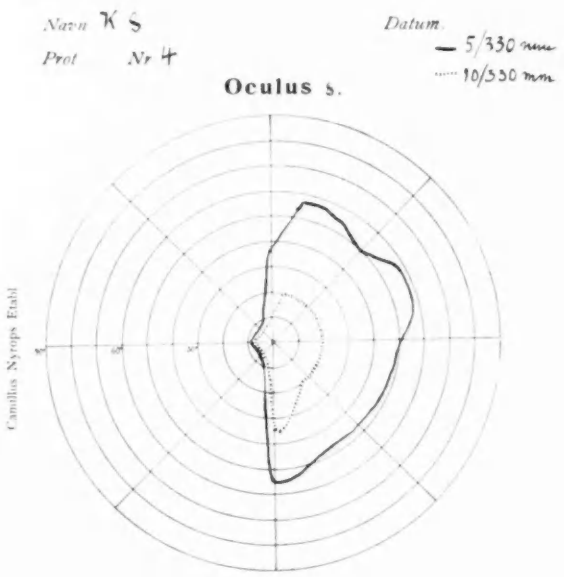
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extremities paraesthesias, later on disturbances of walking. There exists anisocoria (right pupil wider than the left), irregularity of the pupils, Argyll-Robertson syndrome. Light hemiparesis of the left extremities. Insecurity of the finger-nose test, absence of the belly reflexes, patellar and Achilles reflexes. Ataxia of the lower extremities, positive Romberg symptom. Wasserman in the serum negative. Liquor: Wasserman + + +, Pandy + +, Nonne + +, lymphocytes 3, goldsol reaction +. In this period there were found at the second eye clinic: right eye V. = 6/6 with S. — 1.25, left eye V. = 6/6 with S. — 0.5. Left homonymous hemiopia. The left disc paler than the right one. At the nerve department of Prof. Mattauschek, the diagnosis of tabes complicated by a focus in the right hemisphere and left hemiopia was made. The focus causing the hemiopia could not be located. Neither the eye clinic nor the nerve department located it at the base. 1925, attack of iritis. Neosalvarsan was administered at the dermatological department of our hospital. 1927, mercurial treatment.

Examination in January, 1927. Right eye: V. = 10/8 with S. — 1.0. Left eye: V. = 10/8. Right eye: Peripheral corneal nebulæ. Anisocoria (R < L), Argyll-Robertson pupils. Left homonymous hemiopia with overshot field, also with 2/1200 on the Elliot scotometer. The temporal half of the right disc is a little paler than normal, the nasal half of the left disc is distinctly paler than the temporal half. These facts suggested that the case might be one of infranuclear hemiopia. The examination with red-free light showed that the nerve fibers in the right eye are present only in the nasal half of the retina. From the upper margin of the disc the demarcation of the fibers runs vertically upwards; from the lower margin of the disc it slants off about 10° to the nasal side from the vertical meridian. The demarcation between the part of the retina containing nerve fibers and the half without fibers is an absolutely sharp one. Two thin fascicles of nerve fibers pass over the temporal margin of the disc towards the macula. One of them lies immediately above the horizontal meridian of the disc, the other one a little below it. In the left eye, the nerve fibers are missing in the nasal half of the fundus. The demarcation between the part of the retina lacking nerve fibers and the normal one runs vertically from the upper margin of the disc. About corresponding to the middle of the temporal margin of the disc, the fibers going to the macula are completely missing. In the entire area of the papillomacular fascicle the layer of fibers is thinner than normal.

Neither the examination of the field nor the test for hemiopic pupillary reaction could have surely decided the question

of location of the lesion; the field showed with the most minute examination hemiopia with overshot field; the hemiopic pupillary reaction could not be tested on account of the Argyll-Robertson pupil.

The examination in red-free light shows the lack of nerve fibers corresponding to the hemiopia. This is a proof of the infranuclear location of the focus causing the hemiopia, which is a consequence of a lesion of the tractus or the ganglion cells in the second neurone. Examination in red-free light likewise allows us to recognize in each fundus which are the fibers of the papillomacular fascicle that run through the right tract. In the fundus of the right eye they form two groups of fibers above and below the horizon; in the left eye there is a single group of fibers which pass straight from the disc to the macula.

The examination of the fundus in red-free light in the last two cases illustrates the importance of the method for diagnosis. In chiasmal trouble, the condition of the nerve fibers in the retina compared with the defects in the field of vision are of great prognostic significance, namely: if the nerve fibers in the nasal halves of the retinæ are visible, prognosis is good, because the presence of the nerve fibers proves that they are not destroyed, that probably only their conductibility is more or less severely impaired. Treatment in such cases promises not only preservation of function present, but also reëstablishment of function apparently lost. The last case of this group proves the importance of red-free light ophthalmoscopy for differential diagnosis of infra- and supranuclear lesions in homonymous hemiopia. We know that central scotoma can be caused by lesions in different parts of the optic pathways. We can distinguish three principal groups: first, macular lesions; second, central scotoma with focus located in the optic pathways beneath the lateral geniculate body, and, lastly, pathological foci above this center, almost always located in the cortex of the occipital lobe. Red-free light as the method of examination of the retina will allow us to find changes in the macula by actual inspection, or to exclude them on the same grounds. It will, likewise, permit us to ascertain whether there be a lesion of the papillomacular fascicle or not, i.e., whether the focus lies beneath or above the lateral geniculate body. As foci lying in the optic nerve may also be of nasal sinus origin, the differentiation is important for rhinologists.

The close anatomical connection between the retinal and cerebral vessels gives an especial importance to the visible lesions

of the retinal vessels. That vascular changes in the retina are far more distinctly visible in red-free light is shown by the following cases:

Case V

Arteriosclerotic Changes in the Retina

I. Sch., aged 56. Right eye: Externally normal, delicate floating vitreous opacities. Disc of yellowish-pink colour, blurred. Pronounced œdema of the retina in the region of the temporal inferior artery. The main inferior arterial stem appears on the disc as a white cord without visible blood column. Its chief side branches appear as exceedingly thin red lines, which frequently disappear in the gray retina. The temporal inferior artery is thinner than in the normal eye. Its walls reflect light strongly. The inferior temporal vein is as thin as a thread and only feebly visible in the œdematous retina. The temporal superior vein has a normal wide lumen; its branches, however, show great difference of caliber and have occasionally wandering tortuosities. The alterations of caliber in these vessels are so great that the blood column is visible only in some places. The temporal superior artery has but slight coating. There are tiny hæmorrhages in the region of the temporal and nasal arteries. Very small chalky white retinal foci lie on the temporal and upper side of the disc.

In red-free light the changes are much more pronounced than in mixed light. The stem of the inferior artery appears as a white cord and its side branches are accompanied to the distant periphery by white lines. From these white coatings, there branch, in some places, white lines that divide dichotomously without vestiges of blood in them. The superior temporal vein shows also more pronounced vascular changes than in the mixed light. One sees white side-stripes and in some places connective tissue surrounding the vein in a circular way, which strangles it. In the places where the vessels show wandering tortuosities, the accompanying white stripes are especially broad and appear as the chords of the arcs. On the disc a small venous *wondernet (rete mirabile)* is visible, which is perfectly invisible in the mixed light. Also, to the temporal side of the disc, several such *wondernets* become visible which had appeared as hæmorrhages in the mixed light. The hæmorrhages themselves contrast strongly as distinctly outlined black foci with the light background. The yellow macula is distinctly visible; it is only a little paler than usual; it is of normal size; a hæmorrhage covers its temporal margin. The striation of nerve fibers is well preserved on the nasal superior and inferior margin of the disc. On the temporal side they are

scarce and visible only in some places. V. = 5/36 with + 1.0 dptr. cyl. 90°. Only large print is read.

Internal examination: Very pronounced arteriosclerosis with hypertonia; excentric hypertrophy of the left ventricle.

The examination in red-free light grants the advantage of deeper insight into the vascular changes. The wall of the inferior main artery is so highly thickened that the blood column therein contained is not visible. One can deduce surely the presence of endarteritic processes. The conditions of the temporal superior vein, which is visible only in red-free light, are perfectly different. Coatings of connective tissue accompany the vein, surrounding it in a circular manner in some places and strangulate the vessels from the external side. The shrinking of the connective tissue causes the formation of loops and wandering vessels; this is proved by the circumstance that the cords of connective tissue frequently form the chords of the vascular bows. Also, the most delicate *wondernets*, which appeared as hæmorrhages in mixed light or were not visible at all, are distinct in red-free light; from their presence we can conclude that new conditions of circulation have formed. It was not possible in mixed light to recognize surely the localization of the macula; in red-free light it was possible to locate the macula and to find out that its temporal margin is partly covered by a hæmorrhage. The tiniest retinal foci were more distinctly visible in mixed light, because they are set off more distinctly by their bright white colour against the dark red background than against the light background of the fundus seen in red-free light.

Minute hæmorrhages in the retina or the optic disc are far easier to find in red-free than in mixed light. In the following case, the importance of recognition of hæmorrhages for differentiation between pseudoneuritis and papillœdema is clearly demonstrated. This differential diagnosis is, as we all know, sometimes very difficult. If we see a single hæmorrhage in the disc, we will decide for neuritis or papillœdema, because uncomplicated pseudoneuritis will not show any hæmorrhage in the disc or the retina. Consequently, the recognition of minute hæmorrhages will be of the utmost importance.

Case VI

Papilloedema

A. P., aged 54. Since the age of 27, increasing obesity; since a few years, pain in the frontal region; since a month and a half, scintillation before the eyes. Left eye: Disc

reddened, outline blurred, swollen with overhanging borders. S. + .4 dptr. difference of the refraction between the most prominent part of the disc and the level of the retina. Veins choked, tortuous; small hæmorrhages near the temporal inferior vein and near the temporal upper margin of the disc. The rest of the fundus normal.

In red-free light, the tortuosity and passive hyperæmia of the retinal veins is much more striking than in mixed light. In the tissue of the swollen disc there are numerous small radiating hæmorrhages. Coating of the vessels is plainly discernable within the disc. The striation of the nerve fibers is more distinct than under normal conditions and it is visible in the entire fundus.

V. = 10/10. Small print is read. Visual field normal except for enlarged blind spot.

Very pronounced adiposity; no neurological symptoms. Wasserman negative. X-ray examination: Normal sella turcica. No signs of increased intracranial pressure.

The examination proves that the red colouring of the disc is in a great part due to hyperæmia of the smallest vessels and partly to tiny hæmorrhages. Œdema of the papillar tissue has, however, influenced neither the nerve fibers nor the macula. These facts are corroborated by the normal condition of the field with exception of enlargement of the blind spot.

The demonstrated fundus pictures and the explanations applying to them prove sufficiently, I believe, the importance of red-free light ophthalmoscopy not only for the oculist, but also in a high degree for the rhinologist, the neurologist and the internist. Every progress in one of the medical specialties is not only a gain for this specialty, but, at the same time, promotes knowledge and opens new possibilities in many other branches of medicine. How many patients who apply to the oculist for help have been benefited by the progress of rhinology! Let us just remember that operations on the accessory sinuses, and especially on the hypophysis, allow us at present to relieve many patients who, before these operations were given us by our rhinological colleagues, would have had to be left to the sad fate of losing their vision! The improvement of diagnosis by ophthalmoscopy in red-free light, the importance of which for ophthalmology I surely need not dwell upon, proves also useful to our rhinological colleagues. May, in the future, the collaboration of these two branches of medicine, the representatives of which have met here for common work, help to promote further progress of medical knowledge and achievements!

SYMPOSIUM:

THE ETIOLOGY OF EXOPHTHALMOS

CONSTITUTIONAL FACTORS WITH PARTICULAR REFERENCE TO EXOPHTHALMIC GOITER

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ABNORMAL PROTRUSION of the eyeball is encountered in a wide variety of both local and general conditions. Later speakers are to devote attention to those abnormalities, such as hemorrhage and tumor of the nasal and cranial cavities and of the orbit, which by mechanical means produce forward dislocation of the eye. The present discussion is devoted to constitutional diseases in which exophthalmos appears either as a complication or as a part of the general abnormality. A certain amount of overlapping exists because much of the exophthalmos which is encountered in general disease depends on the local accumulation in the orbit of tumors, hematomas, or other masses; or on the production of stasis in the orbital veins; or on edema of the orbital tissues; all of which may result in forward dislocation of the eyeball. This generalization at times applies to the exophthalmos of exophthalmic goiter, which will be considered presently. It is partly, often wholly, applicable to the exophthalmos that occurs occasionally in other constitutional abnormalities as follows:

EXOPHTHALMOS IN DISEASES OTHER THAN EXOPHTHALMIC GOITER

Exophthalmos is very rare in syphilis. Delord described bilateral protrusion of the eyes that occurred in the clinical course of a lesion of the pituitary body which was probably of

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syphilitic nature, and Löhlein wrote of a paretic exophthalmos that appears in the course of syphilis or tabes, when paralysis of the external muscles of the eye occurs. This is never of high grade and is accounted for by the loss of restraining action of the external ocular muscles. Gumma may involve the orbit, and there may be a tubercle of the orbit in tuberculosis; other granulomas may occur in a similar manner. Key recently wrote of a case in which exophthalmos accompanied trichinosis, and Holmström described the cases of two sisters with exophthalmos of the right eye, which occurred in neurofibromatosis (Recklinghausen's disease). A few cases of this have been seen by Benedict at The Mayo Clinic.

Exophthalmos is encountered also in cases of multiple xanthomatosis. This is a generalized disease in which tumorous masses of reticular cells are engorged with lipids, predominantly cholesterol. Occasionally, the condition is associated with diabetes insipidus and defects of the membranous bones. It then constitutes the so-called Schüller-Christian syndrome. Multiple xanthomatosis is rare and exophthalmos is, by no means, an invariable accompaniment of it. We have recognized only one instance of xanthomatosis with exophthalmos.

The various forms of leukemia sometimes are responsible for proptosis, but Giffin, with his large experience in diseases of the blood, could tell us of no single instance at the clinic, and the literature contains very little about the condition. The only lymphoid tissue in the orbit, according to Reese and Guy, is situated under the bulbar conjunctiva and in the lacrimal gland. The bulging of the eye, in a case of lymphatic leukemia that was described by these authors, was the result of swelling of the conjunctiva from the accumulation beneath it of a mass of lymphocytes. Exophthalmos in myelocytic leukemia is almost unknown. In the one case that was seen by Reese and Guy, enucleation of the eyeball disclosed that the orbit was filled with blood. An instance of exophthalmos, which occurred in monocytic leukemia, was recently reported by Mesropyan.

Hemorrhage in the orbit is the cause of the proptosis that is an occasional complication of hemophilia, rickets, and scurvy. According to Löhlein, bilateral subperiosteal hematomas in the orbit, with the resulting exophthalmos, for a long time may be the only manifestation of rickets. Raines wrote that approximately 2,000 children, aged from two weeks to two years, make

15,000 calls yearly to the infirmary in the Russian city, Astrakan; in 1928, 620 out of 2,020 children were obviously rachitic, and of these, 120 had exophthalmos. There was a positive correlation between this symptom and the severity of the rickets, and with the disappearance of the rickets, it disappeared. The exophthalmos of rickets differs from that of exophthalmic goiter in that the lower lid is edematous and hangs down in the middle, and the upper lid, although also swollen, is not retracted. It may be mentioned in this connection that Judkin obtained exophthalmos, among other abnormalities, in rats that had been fed diets that were low in mineral elements.

Protrusion of the eyeball was discovered in nearly 10 per cent of 379 cases of scurvy that were investigated by the American Pediatric Society, in 1898. This is quoted from an article published in 1931 by Dunnington, who commented, however, that De Buys, in 1912, had been able to discover only twelve reported cases of scurvy in which the evidence justified, in his opinion, the diagnosis of proptosis. To these twelve cases, Dunnington added ten more, including one of his own. He explained the condition of the eye, in all instances, on the basis of hemorrhage, usually subperiosteal, of the roof of the orbit. The protrusion of the eyeball is forward, downward, and outward; it is of varying intensity and is usually a late manifestation. The average age of the patients is twenty-two months, but Krükow recorded the case of an adult, seen in Russia, who had scurvy and exophthalmos. The exophthalmos receded rapidly as a result of treatment of the scurvy.

The occurrence of exophthalmos in hypertension and nephritis is a subject to which we have devoted considerable attention since it was reported by Barker and Hanes in 1909. Their conclusions, which were supported by Thayer, left the impression that it was a frequent complication of this condition. It should be noted that many of their patients were negroes, a race in which prominent eyes are not unusual, that no exophthalmometer readings were reported, and that the possibility of the coexistence of exophthalmic goiter may not have been excluded as completely as it would be excluded today. In any event, our experience is not the same. The determination of the presence or absence of exophthalmos and the estimation of degrees of proptosis in exophthalmic goiter, as well as the differential diagnosis of true exophthalmos and of conditions which simulate it, have been

among our major problems for many years. It is our opinion, as well as that of H. S. Plummer and his other associates, that retraction of the lids and possibly other factors which may contribute to a facial expression somewhat like that observed in exophthalmic goiter, are not infrequent in nephritis, but that the occurrence of proptosis is extremely rare. The difficulty of distinguishing eyes that are affected with true exophthalmos from those that are affected simply by retraction of the lids is exemplified in certain cases of exophthalmic goiter; one eye may seem to be more prominent than the other, when the exophthalmometer readings are identical, and the apparent prominence is entirely attributable to retraction of the lids.

Confusion on this score seems to be responsible for most of the diagnoses of exophthalmos that are reported, not only in hypertension and nephritis, but also in other conditions of so-called sympathicotonia. Similarly, the coexistence of exophthalmic goiter must frequently have escaped recognition. Conditions of mild hyperthyroidism easily are overlooked, and patients who have exophthalmic goiter and who have no evidences of hyperthyroidism, at the time of observation, frequently are seen, and under these circumstances the condition is difficult to recognize. This has been emphasized by H. S. Plummer.

EXOPHTHALMOS IN EXOPHTHALMIC GOITER

Exophthalmic goiter, in which there is associated diffuse hyperplasia of the parenchymal epithelium of the thyroid gland, is the one constitutional condition in which exophthalmos is truly significant. Therefore, conclusions regarding the etiology and mechanism of the exophthalmos of constitutional diseases must depend primarily on the information that is accumulated by the study of this disease.

EXPERIMENTAL EXOPHTHALMOS

In 1858 and 1859, Heinrich Müller described the presence of smooth muscle in the eyelids. Landström said that, as late as 1901, no smooth muscle had been recognized within the orbit, and that Merkel and Kallius, writing in that year for the Gräfe-Samische Handbuch, insisted that the muscles of Müller extended only from the orbital margin to the tarsus and thus were in no way able by their contraction to influence the position of the bulb. Landström, with serial sections of entire orbits in three

perpendicular planes, was able to demonstrate a cone of smooth muscle that extended from the orbital margins to a position behind the equator of the eyeball. He attributed to this muscle the rôle of an antagonist to the four rectus muscles, whereby the axis of the eye was given proper stability. The observation on animals, originating with Claude Bernard, that stimulation of the cervical sympathetic nerves caused forward displacement of the eyeball, Landström considered to be explained by the presence of this cone of smooth muscle. He emphasized that these muscle fibers are overlooked if the eye is enucleated, because they then remain behind in the orbit. Serial sections of the entire orbit are necessary for their demonstration.

MacCallum and Cornell likewise discovered that the smooth muscle fibers of Müller extended backward as a conical mantle about the eye. While they were more fully developed in the dog, they were by no means wanting in man. These investigators removed the roof of the orbit in dogs and by electrically stimulating the cervical sympathetic nerves, they could observe a distinct peristaltic wave, which passed backward through the length of this cone and pressed the eyeball forward into the position in which it is seen in exophthalmos. When the membranous cone was snipped with scissors and the cervical sympathetic nerves were stimulated, the eyeball remained stationary. Joumesco reported the production of exophthalmos at *surgical operation*, by stimulating the sympathetic nerves with a strong current, but in two patients that MacCallum and Cornell were able to observe, through the courtesy of the surgical staff of Johns Hopkins Hospital, stimulation of the cervical sympathetic nerves did not produce visible exophthalmos, although the pupil became greatly dilated on the stimulated side.

This anatomic and experimental evidence relative to the influence of Müller's or Landström's muscle is accepted as a satisfactory explanation of the mechanism of exophthalmos by the writer of an editorial that appeared recently. The complexity of the clinical problem is ignored. No such mechanism can possibly produce the more extreme grades of proptosis which are encountered in some cases of exophthalmic goiter, and surgeons who are acquainted with the anatomy of the human eye are almost universally of the opinion that the smooth muscle fibers of the orbit are developed too poorly in many, if not in most, instances to affect the position of the eyeball. Further-

more, a widening of the pupil always accompanies artificial stimulation of the cervical sympathetic nerves and is seen clinically in irritative lesions of the neck; whereas, this is, at least, very unusual in the exophthalmic eye of exophthalmic goiter. The careful measurements of the pupils in a small series of cases which have beeen reported by Holloway, Fry and Wentworth, conform to this conclusion.

Possibly the absence of mydriasis in the exophthalmos of exophthalmic goiter has been explained by the recent studies of Labbé and his associates. Briefly, their results were as follows: Ephedrine when injected into dogs in doses of 0.5 mg. per kilogram produced mydriasis and exophthalmos that were scarcely noticeable; whereas, if thyroxin was administered either before or afterward, intense exophthalmos developed without mydriasis. There was no intraocular increase in tension, no engorgement of vessels, and no edema or retrobulbar congestion.

Epinephrine, when administered with thyroxin, had a like effect. This, we may add, was noted also by Kendall and Haines in early unpublished experiments with thyroxin, after its isolation by Kendall.

The following clinical experiments are described by Labbé: An obese woman was receiving an enormous dose of thyroxin by mouth (10 mg. a day) and marked signs of hyperthyroidism were in evidence. The patient was given 50 mg. of ephedrine; five minutes later striking exophthalmos appeared, which was more marked in the left eye. It attained its maximum in ten minutes and persisted for two hours. It was not accompanied by the slightest degree of mydriasis. Subsequently, two patients who had exophthalmic goiter but no exophthalmos, and who had basal metabolic rates of about +60 per cent, were given ephedrine, and exophthalmos developed and attained a degree of considerable importance within an hour and a half. This likewise was unaccompanied by mydriasis.

Exophthalmos that was accompanied by miosis was produced, in animals, with various combinations of miotic poisons and sympathomimetic drugs, and the assumption was made that thyroxin not only stimulates the sympathetic nerves but also exerts miotic action by simultaneous stimulation of the parasympathetic mechanism, whereby its mydriatic effects are neutralized.

These observations, if confirmed, will constitute an important contribution. It is unfortunate that there is no statement,

in these reports, regarding actual measurements of the degrees of exophthalmos. It should be mentioned that Labbé and his associates also discovered that yohimbine was antagonistic to ephedrine and epinephrine, and tended to counteract the exophthalmos that was produced in animals by the latter drugs, and that in some cases it exerted a favorable influence on the exophthalmos of exophthalmic goiter.

These authors, as well as others who have experimented with animals, have been unable to provoke exophthalmos with thyroxin alone, even when the doses are sufficiently large to produce tachycardia and loss of weight. However, Kunde reported positive results with this procedure. Dogs, she wrote, are refractory to thyroxin; but in normal rabbits, a mild exophthalmos is obtained; while in rabbits, which previously have been rendered myxedematous by thyroidectomy, it develops to a high degree.

Exophthalmos has also been produced in animals by injection of extracts of the anterior lobe of the pituitary body. It is now well established that ablation of the pituitary body causes atrophy of the thyroid gland and a significant decrease in the basal metabolic rate, and that injection of extracts of the anterior lobe of the pituitary body stimulates temporarily the activity of the normal thyroid gland. The latter phenomenon is accompanied invariably by hypertrophy of the parenchymal epithelium of the thyroid gland. The injections also are followed by exophthalmos, as was noted first in ducks by Schochaert, and as was noted later in guinea pigs by L. Loeb and Friedman, by Marine and Rosen, and by Friedgood. Marine and Rosen expressed the opinion that exophthalmos could be produced even in the absence of the thyroid gland. In Friedgood's experiment, in nine of thirty guinea pigs that were given injections of extract of the anterior lobe of the pituitary body, exophthalmos of obvious degree developed; at one time or another this exophthalmos was essentially unilateral. When it was bilateral, one eye was frequently more prominent than the other.

A feature of the hyperthyroidism that is produced by repeated daily injections of extracts of the anterior lobe of the pituitary body is that it is transient. Loss of weight and other evidence of increased activity of the thyroid gland appear for ten or fourteen days and after this, the animal enters a refractory stage of apparent immunity. In a few of Friedgood's animals,

the exophthalmos was observed by the tenth day, that is, during the height of the hyperthyroidism; in others it did not appear until after the refractory stage had been reached. The most striking exophthalmos was seen in those animals that were given injections over a period of several months, and especially in the animals in which abnormally low basal metabolic rates finally were obtained. Chronic exophthalmos, which persisted after the injections had been discontinued, resulted in some cases.

These last instances of exophthalmos are extremely interesting; they are analogous, as will appear, to the exophthalmos that occurs in cases of remission of exophthalmic goiter. The most severe ocular complications that lead to so-called malignant exophthalmos in exophthalmic goiter develop after thyroidectomy has been performed, or after spontaneous remission of the symptoms, when the basal metabolic rate has fallen to normal or even to less than normal. However, the evidence deduced from experiments on animals must be applied to the human being with the greatest caution. Exophthalmic goiter does not develop spontaneously in animals, as it does in man, and in our enthusiasm over the experimental production of exophthalmos, we must not disregard the tremendous amount of evidence provided by clinical experience. One fact, above all others, must be held in mind; namely, that resection of the thyroid gland, in the vast majority of instances of exophthalmic goiter, not only brings about subsidence of the symptoms of hyperthyroidism but also recession, or at least standstill, of any previous exophthalmos. This demonstrates that the process, which is responsible for the development of exophthalmos in this disease, is, in most cases, markedly interfered with, if not entirely inhibited, by removing the influence of the thyroid gland. It is only exceptionally that exophthalmos progresses when other evidence of overactivity of the thyroid gland has disappeared, and too much emphasis should not be placed on these exceptions. The vast amount of clinical evidence still indicates that the immediate cause of exophthalmos in exophthalmic goiter is closely linked with actual changes in the function and pathology of the thyroid gland.

CLINICAL OBSERVATIONS

This brings us to the clinical data at hand. The incidence of exophthalmos in exophthalmic goiter is variously reported by different authors. In Joll's series, it was 74 per cent; Boothby

reported it to be 65 per cent; and Barker estimated it at only 34 per cent. We have reviewed the records of 2,000 patients at the clinic, in an attempt to correlate the presence or absence of exophthalmos, and its degree, with the duration of the disease and with certain clinical features which are believed to be related to the severity of the disease and its duration. These are, namely, the basal metabolic rate, and the loss of strength of the quadriceps femoris muscles. H. S. Plummer has called attention to the variations in the intensity of exophthalmic goiter that occur from time to time, at least in certain localities; periods of increased severity may last a year or more and alternate with periods of relative mildness. Also, a large percentage of patients who have exophthalmic goiter and who have been seen in recent years, have been treated with iodine, and this has modified the severity of the symptoms and has lowered the basal metabolic rate of those who have been treated just previous to coming under observation. Consequently, it seemed advisable to study two groups of cases, removed from each other in point of time, taking 1,000 before 1922, and an equal number after 1930. The cases in each group are consecutive, but only those patients who had undergone previous thyroidectomy are included; the diagnosis of exophthalmic goiter, therefore, has been established not only by the clinical evidence but also by direct examination of the thyroid gland and by the pathologic finding of diffuse hypertrophy of the parenchymal epithelium. Some of the thyroid glands contained adenomas, but no cases belonging to the clinical entity known as adenomatous goiter with hyperthyroidism have been included. Exophthalmos is not an accompaniment of adenomatous goiter with hyperthyroidism. For all practical purposes, it is limited exclusively to the entity known as exophthalmic goiter.

The data are as follows: Table 1 contains the basal metabolic rates. It reveals that the average severity of hyperthyroidism was relatively lower for the later group of cases, + 42.4, than it was for the earlier group, + 53.5.

Table 2 gives the distribution of patients by age and by sex. The mean age was slightly higher in the later period. This is true both for males and females. The proportion of males is appreciably higher in the later group.

Table 3 reveals a positive correlation of basal metabolic rate and relative incidence of exophthalmos. For patients who

TABLE 1

*Basal metabolic rate in exophthalmic goiter**

B. M. R. Per cent	BEFORE 1922 Patients	BEFORE 1922 Per cent of Patients	AFTER 1930 Patients	AFTER 1930 Per cent of Patients	TOTAL Patients	TOTAL Per cent of Patients
—30 to —20			1	0.1	1	0.05
—20 to —10						
—10 to 0			3	0.3	3	0.15
0 to 10	5	0.5	14	1.4	19	0.95
10 to 20	31	3.1	79	7.9	110	5.50
20 to 30	91	9.1	181	18.1	272	13.60
30 to 40	156	15.6	203	20.3	359	17.95
40 to 50	166	16.6	169	16.9	335	16.75
50 to 60	193	19.3	167	16.7	360	18.00
60 to 70	131	13.1	94	9.4	225	11.25
70 to 80	107	10.7	51	5.1	158	7.90
80 to 90	64	6.4	23	2.3	87	4.35
90 to 100	39	3.9	10	1.0	49	2.45
100 to 110	11	1.1	3	0.3	14	0.70
110 to 120	1	0.1	2	0.2	3	0.15
120 to 130	1	0.1			1	0.05
130 to 140	2	0.2			2	0.10
140+	2	0.2			2	0.10
TOTAL	1000	100.0	1000	100.0	2000	100.00
Mean B.M.R.	+53.5		+42.4		48.0	

* The mean basal metabolic rate is lower after 1930 than it was before 1922.

TABLE 2

*Distribution of patients by age and sex**

	PRIOR TO 1922 Patients	PRIOR TO 1922 Per cent	PRIOR TO 1922 Mean age, years	AFTER 1930 Patients	AFTER 1930 Per cent	AFTER 1930 Mean age, years	TOTAL Patients	TOTAL Per cent	TOTAL Mean age, years
Males	225	22.5	39.3	374	37.4	42.7	599	30.0	41.4
Females	775	77.5	35.0	626	62.6	40.4	1401	70.0	37.4
TOTAL	1000	100.0	36.0	1000	100.0	41.3	2000	100.0	38.6
Sex ratio female to male	3.4			1.7			2.3		

* The mean age of patients who had exophthalmic goiter was higher in the period after 1930 than it was in the period before 1922. This is true both for males and females. The ratio of females to males was greater in the earlier period (3.4) than it was in the later period (1.7).

TABLE 3

*Relation basal metabolic rate to exophthalmos — 2000 cases**

	EXOPHTHALMOS											
	Absent		Grade 1		Grade 2		Grade 3		Grade 4		Present but not graded	
B. M. R.	Patients	Per cent	Patients	Per cent	Patients	Per cent	Patients	Per cent	Patients	Per cent	Patients	Per cent
—30 to —20					1	0.3						
—20 to —10												
—10 to 0	2	0.2			1	0.3						
0 to 10	8	0.9	3	0.6	4	1.1	3	2.9			1	**1.3**
10 to 20	54	5.9	22	4.2	20	5.4	5	4.9	1	11.1	7	8.9
20 to 30	155	17.1	68	13.0	35	9.5	4	3.9	1	11.1	8	10.1
30 to 40	195	21.5	78	14.9	54	14.6	20	19.6			11	13.9
40 to 50	158	17.4	93	17.8	52	14.1	17	16.7	2	22.2	12	15.2
50 to 60	145	16.0	110	21.0	66	17.9	18	17.6	3	33.3	16	20.2
60 to 70	103	11.3	64	12.2	40	10.8	10	9.8	1	11.1	7	8.9
70 to 80	55	6.1	42	8.0	39	10.6	13	12.7			**8**	**10.1**
80 to 90	16	1.8	24	4.6	31	8.4	8	7.8			6	7.6
90 to 100	9	1.0	11	2.1	21	5.7	4	3.9	1	11.1	3	3.8
100 to 110	6	0.7	6	1.1	2	0.5						
110 to 120	2	0.2			1	0.3						
120 to 130					1	0.3						
130 to 140			2	0.4								
140+	1	0.1			1	0.3						
TOTAL	909	100.0	523	100.0	369	100.0	102	100.0	9	100.0	79	100.0
Average B.M.R.	44.1		50.0		53.1		51.5		50.4		49.8	

* There appears to be a positive correlation of basal metabolic rate and relative incidence of exophthalmos. For patients who had no exophthalmos, the average basal metabolic rate was +44.1; for those who had exophthalmos in various degrees, the basal metabolic rate was +50 or more.

TABLE 4

*Percentage frequency of exophthalmos in exophthalmic goiter**

	PRIOR TO 1922		AFTER 1930		TOTAL	
	Patients	Per cent	Patients	Per cent	Patients	Per cent
Exophthalmos	689	68.9	402	40.2	1091	54.5
No exophthalmos	311	31.1	598	59.8	909	45.5
TOTAL	1000	100.0	1000	100.0	2000	100.0

* The percentage frequency of exophthalmos in exophthalmic goiter was greater before 1922 than it was after 1930. Almost 70 per cent of the earlier group had exophthalmos; only 40 per cent in the later group had exophthalmos.

TABLE 5

*Percentage frequency of grades of exophthalmos in exophthalmic goiter**

Exophthalmos (D.O.D.)	PRIOR TO 1922 Patients	Per cent	AFTER 1930 Patients	Per cent	TOTAL Patients	Per cent
None	311	31.1	598	59.8	909	45.5
Grade 1	337	33.7	186	18.6	523	26.2
Grade 2	225	22.5	144	14.4	369	18.4
Grade 3	78	7.8	24	2.4	102	5.1
Grade 4	5	0.5	4	0.4	9	0.4
Present, but not graded	38	3.8	41	4.1	79	4.0
Left eye only	6	0.6	3	0.3	9	0.4
TOTAL	1000	100.0	1000	100.0	2000	100.0

* The greater percentage frequency of exophthalmos in the earlier period is revealed for all grades of exophthalmos.

had no exophthalmos, the average basal metabolic rate was + 44; for those who had exophthalmos in various degrees, the basal metabolic rate was + 50 or more.

The percentage frequency of exophthalmos in exophthalmic goiter was definitely greater before 1922 than it was after 1930 (table 4). This correlates with the lower basal metabolic rates that were discovered in the later group. It is apparent for all grades of exophthalmos (table 5).‡ In other words, in the group which had the lower basal metabolic rates, the incidence of exophthalmos was also lower.

Table 6 reveals a positive correlation between the duration of hyperthyroidism and the relative frequency of exophthalmos. In the entire 2000 cases of exophthalmic goiter there is a frequency of 48.7 per cent exophthalmos in the cases of less than six months' duration, and this rises steadily to about 60 per cent for the cases having a duration of two to four years and to 71.2 per cent for cases over four years.

In the period before 1922, the percentage frequency of exophthalmos in the cases showing no loss of quadriceps strength was 62.7 per cent; this frequency increased steadily to 77.6 per

‡ The grading of exophthalmos in these cases was based on inspection alone. The examiners, however, were physicians who had been trained to distinguish true prominence of the bulb from apparent prominence resulting from retraction of the lids. There is no reason to believe that the percentage of error in series as large as these would change; therefore, the grading that was done after 1930 can be assumed to be the same as the grading that was done before 1922.

TABLE 6

*Relation of duration of constitutional symptoms of exophthalmic goiter to exophthalmos**

Constitutional Symptoms	PRIOR TO 1922			AFTER 1930			TOTAL CASES		
	Patients	Exophthalmos Cases	%	Patients	Exophthalmos Cases	%	Patients	Exophthalmos Cases	%
0 to 5 mo.	241	164	68.0	262	81	30.9	503	245	48.7
6 to 11 mo.	196	136	69.4	166	73	44.0	362	209	57.7
1 to 2 yr.	195	128	65.6	136	67	49.3	331	195	58.9
2 to 3 yr.	85	58	68.2	68	35	51.5	153	93	60.8
3 to 4 yr.	36	28	77.8	30	11	36.7	66	39	59.1
4 yr. +	87	68	78.2	69	43	62.3	156	111	71.2
Not stated	160	107	66.8	269	92	34.2	429	199	46.4
TOTAL	1000	689	68.9	1000	402	40.2	2000	1091	54.6

* There is a positive correlation indicated between the duration of the constitutional symptoms of exophthalmic goiter and the percentage frequency of exophthalmos.

TABLE 7

*Relation of loss of strength of quadriceps femoris muscles to exophthalmos**

Loss of Quadriceps Strength	PRIOR TO 1922			AFTER 1930			TOTAL CASES		
	Patients	Exophthalmos Cases	%	Patients	Exophthalmos Cases	%	Patients	Exophthalmos Cases	%
None	83	52	62.7	142	48	33.8	225	100	44.4
Grades 1 and 2	424	289	68.1	465	184	39.6	889	473	53.2
Grade 3	178	130	73.0	104	50	48.1	282	180	63.8
Grades 4 and 5	58	45	77.6	67	30	44.7	125	75	60.0
Not stated	256	173	67.6	210	86	41.0	466	259	55.6
Present, but not graded	1	0	0	12	4	33.3	13	4	30.8
TOTAL	1000	689	68.9	1000	402	40.2	2000	1091	54.6

* There is a definite positive correlation between the degree of the loss of quadriceps strength and the presence of exophthalmos.

cent as one went through the groups up to grade 5 loss of strength. In the period after 1930 the percentage frequency in the cases showing no loss of strength was 33.8 per cent, and this rose to 44.7 per cent in cases with grade 5 strength loss.

This table also indicates that among the patients who had been examined before 1922, exophthalmos had developed earlier

in the course of their disease than among those who had been examined after 1930.

Loss of strength of the quadriceps femoris muscles, as estimated by the ability of the patient to step up on the step of the examining table, has been recorded for many years. Table 7 discloses a positive correlation between the degree of the loss of strength of the quadriceps femoris muscles and the presence of exophthalmos.

The bearing of this correlation on the mechanism of exophthalmos is to be considered. It rests on the assumption that loss of strength of the quadriceps femoris muscles is paralleled by a similar loss of strength of the external ocular muscles, and that the loss of tonus of the external ocular muscles predisposes to forward dislocation of the eyeball.

The statistics make it clear that, for large groups of patients, the presence or absence of exophthalmos is correlated directly with the severity of hyperthyroidism, as measured by the basal metabolic rate and the presence of weakness of the quadriceps femoris muscles; also, that after 1930, when the severity of exophthalmic goiter was, on the whole, milder than it was before 1922, the incidence of association diminished significantly.

A great many writers have emphasized rapid fluctuation in exophthalmos, in individual instances, and several of them have used this as an argument to support the theory that irritation of the cervical sympathetic nerves is the determining factor in the condition; they maintain that edema or other gross anatomic alteration could not produce such rapid variations. This, however, is certainly incorrect. There is variation, but it is very slow and not apparent from day to day, as has been so frequently written in both old and recent literature. The other ocular symptoms vary, especially the width of the palpebral fissure (Stellwag's sign), the retraction of the upper eyelids (Dalrymple's sign), and the lag of the lid (Graefe's sign); but measurements with the exophthalmometer, which are taken frequently on the same patient, indicate that any change in actual exophthalmos is insignificant, except as there may be a gradual increase or decrease extending over weeks, months, or years.

The exophthalmos that accompanies the active stages of exophthalmic goiter is almost always moderate in degree, as appears from the tables. As a rule, it is bilateral and equal, although

in many cases there will be an appearance of asymmetry, which may or may not be due to a difference of 1 or 2 mm. of exophthalmos. It occasionally is accompanied by weakness of the external ocular muscles, but strabismus is uncommon. It is accompanied by more or less marked spasm of the muscles of the lids but there is seldom more than very slight edema of the lids and conjunctivæ, and very rarely are there serious ocular complications, such as corneal ulcer and panophthalmitis. When exophthalmos is present, its clinical course usually parallels the increase or decrease in the severity of the exophthalmic goiter; in a large majority of cases, the eyes gradually return to normal during the remissions which follow thyroidectomy or occur spontaneously. This is particularly true of the lower grades of exophthalmos. Exophthalmos is affected to a great extent by its previous duration and by its intensity. Some residual exophthalmos usually remains after proptosis of a high degree.

EXOPHTHALMOS IN EXOPHTHALMIC GOITER WITH LOW BASAL METABOLIC RATE

The clinical data which have been presented were obtained from patients who had had no previous operation on the thyroid gland, but the course of exophthalmos in so-called recurrent exophthalmic goiter is no different. After a second resection of the thyroid gland, exophthalmos, if present, usually recedes again, and although some degree of residual proptosis may remain in cases in which it was most marked, further progression is arrested.

However, there is a group of unusual cases in which exophthalmos, which previously has been absent, first develops after a thyroidectomy, or having been present before, increases afterward, even though the constitutional symptoms of the disease have abated and the basal metabolic rate has fallen to normal or even so far below normal as to provoke evidences of myxedema. In such cases, resection of additional thyroid tissue is seldom of any benefit. Comparable to these, there is a small number of cases in which exophthalmos develops after a spontaneous remission from a previous elevation of the basal metabolic rate, and there is a still smaller number in which it may develop in the absence of any evidence of preceding hyperthyroidism. We have seen a few instances of each of these conditions. The clinical features which are common to all cases, are a basal met-

abolic rate that is normal or lower than normal, and a tendency to progess to severe degrees of proptosis, which is associated with marked edema of the lids. It is in this group that the vast majority of instances of so-called malignant exophthalmos develop.

Zimmerman described eight instances of the abnormality among patients who previously had undergone thyroidectomy. He called the condition "paradoxical exophthalmos." Three of the patients had had no ocular abnormalities, three had had slight exophthalmos, and two others had disclosed a Graefe's sign at the time of the thyroidectomy. In all of them the severe proptosis developed after the operation and was accompanied by edema of the lids, chemosis of the cornea, and other more serious complications. In three of them, the disturbance was limited to one eye; in the remainder, it was bilateral. The basal metabolic rates in three were below —14, and evidences of hypothyroidism were present in four. Thompson previously had reported three patients who had had acute edema of the orbit, which developed after thyroidectomy; in one case it necessitated enucleation of both eyes. Zimmerman considered it altogether possible that such edema may have been the factor responsible for the proptosis that was present in his cases. Two of the patients in Thompson's group had undergone thyroidectomy at the clinic, and our records disclose that the proptosis, at the time of thyroidectomy, was graded 3+ in one, but only 1+ in the other.

Haines has described a case in which symptoms of exophthalmic goiter, including exophthalmos, were superimposed on those of myxedema, which followed thyroidectomy. Burch has described a case in which a patient had progressive exophthalmos and a low basal metabolic rate, and Roeder has added seven cases in which the patients had low basal metabolic rates; three of these patients had not had previous thyroidectomy. Roeder has directed attention to the frequency of accompanying paralysis of the external ocular muscles. Interference with the function of the superior rectus muscles, which results in inability to elevate one or both eyeballs, is the usual abnormality, but we have secured photographs of two patients who had internal strabismus, which was the result of paralysis of the external rectus muscles. King has added five cases in which patients who had exophthalmos also had low basal metabolic rates; and Naffziger has added six similar cases.

PATHOLOGIC OBSERVATIONS

It is from the specimens, which have been obtained from orbital explorations, enucleations, and decompressions in cases of the type in question, that most of our recent knowledge about the orbital content in the exophthalmos of exophthalmic goiter has been derived. This knowledge obviously applies only to this relatively infrequent and peculiar condition and may not be correctly informative as to the nature of the exophthalmos that occurs in the stages of exophthalmic goiter that are associated with elevated basal metabolic rates. The information that has been obtained from the small group of patients who had malignant exophthalmos is as follows:

Benedict, while exploring the orbits of four patients who had extreme proptosis, was impressed by the fact that all the tissues were waterlogged. There was no excessive fat in the orbit. The patients had basal metabolic rates that were normal or lower than normal. Two of these patients had undergone thyroidectomy. Another patient had not undergone thyroidectomy, although the diagnosis of exophthalmic goiter was definite; the determinations of the basal metabolic rates, at the time of the enucleation of the eye, were + 3 and + 11. Foster Moore, while dissecting "excessive fat" from the orbit of a patient who had exophthalmos, discovered that the muscles were greatly swollen. "Instead of being thin, flat, ribbon-like structures, such as one becomes familiar with in squint operations, they had fusiform bellies not quite so stout as the last joint of one's little finger." The case of Burch, which already has been referred to, was that of a man, aged sixty-two years. Three years previously, when he was examined at the clinic, he had a "stare" and a Graefe's sign, but he had no definite exophthalmos. The basal metabolic rate was determined twice; the first time it was + 20, and at another time it was + 30. The thyroid gland, which was removed, disclosed hypertrophied epithelium. The recovery from the thyroidectomy was complete; he remained well until 1928, then exophthalmos developed. Ultimately, both eyes had to be enucleated, and the muscles that surround, and are attached to, the orbit were found to average 1 to 3 cm. in thickness. A cross-section disclosed muscle fibers which were in various stages of atrophy and which were surrounded by loose fibrous tissue and edema. This seems to be the first detailed description of a peculiar pseudohypertrophy of orbital muscles, which, to judge

from the subsequent reports of Naffziger, may act as a tumor in the production of exophthalmos. The very complete reports of Naffziger deal with six cases in which he accomplished decompression of the orbit by his original operation. In this procedure the orbital plate of the frontal bone is resected. There was enormous hypertrophy of the rectus muscles in every instance. They were several times their normal thickness and were of an unusual rubbery firmness. There was some interstitial edema and proliferation of lymphocytes. At necropsy in a similar case at the clinic, which is to be reported later, the orbits were disclosed to be literally packed with enlarged rectus muscles; this condition was similar to that described by Naffziger. There was also congestion of the vessels and considerable edema, which involved all the orbital tissues. The orbital fat was edematous, but it was not excessive in amount.

A satisfactory explanation for this type of exophthalmos is difficult to discover. Mention has been made of the somewhat similar observations by Marine, Rosen, Friedgood, and Kunde, in experimental exophthalmos. The animals that had no thyroid glands, or those that had low basal metabolic rates were those in which the most extreme grades of proptosis developed under the influence of the various agents employed. The possibility must be held in mind that some influence on the part of the pituitary body may cause human beings to be affected in a similar manner, but there is no clinical evidence to warrant such a conclusion. Another explanation is provided by the well-known two-product theory of H. S. Plummer. An abnormal product secreted by the diseased thyroid gland is responsible, according to this theory, for the exophthalmos in exophthalmic goiter, but not for the elevation of the basal metabolic rate. The latter depends on the quantity of thyroxin, or normal product, which is supplied by the thyroid gland. Now, the continued activity of the remnant of thyroid gland that remains after thyroidectomy, might, under certain circumstances, supply enough of the abnormal product to provoke progression of the exophthalmos and yet not be able to supply enough thyroxin to maintain the basal metabolic rate. It would thus provoke exophthalmos in the presence of basal metabolic rates that are no higher than those disclosed in cases of myxedema. Finally, there is some reason to believe that the peculiar edematous infiltration of the structures of the orbit, which occurs with these extreme

degrees of exophthalmos, and which develops in patients who have low basal metabolic rates, is like the edematous infiltration which occurs infrequently as localized plaques of solid edema in the cutaneous and subcutaneous tissues of the head, trunk, and extremities. The instances in which this has been observed are few, and in all of eight cases, which have been reported by O'Leary, the patients had undergone thyroidectomy, which was followed by varying periods of remission of the symptoms of exophthalmic goiter. The improvement, in turn, had been succeeded by the development of the plaques. The records of these cases bear a close resemblance to those of the cases of exophthalmos that are associated with low basal metabolic rates, and in some instances exophthalmos has been an associated symptom. It is possible that a similar process, which involves the orbital structures, may account for exophthalmos in exophthalmic goiter with a low basal metabolic rate.

When the investigator turns to the consideration of exophthalmos, as it commonly occurs in association with elevated basal metabolic rates and constitutional symptoms of hyperthyroidism, information is meager. Basedow, Möbius, and other older writers believed that an excessive amount of fat in the orbital cavity was an important factor, but this does not seem to have been supported. There is no positive evidence that excessive amounts of fat ever have been discovered in the orbital cavity. A feature of exophthalmic goiter is the disappearance of body fat, and it would be strange, indeed, if fat persisted selectively in the orbit. It is possible that edematous infiltration of the orbital tissue has been mistaken for an increase in the amount of fat. A more plausible theory is the one which maintains that edema is responsible for protrusion of the eye, but this is objected to on the ground that accumulation of much fluid in the orbit would be accompanied by more swelling of the lids than ordinarily is seen. Mechanical theories, which depend principally on the assumption that engorgement of the vessels is necessary for the production of edema, also have been advanced by King, O'Day, and others.

It is difficult to discard the theories based on stimulation of the sympathetic nerves. The effect of sympathectomy in overcoming retraction of the lid in cases of exophthalmic goiter is undoubted, and in some cases proptosis also has been affected. The experiments of Labbé and his associates, which relate to this question, have been referred to.

There is a dearth of protocols of postmortem examination bearing on the content of the orbits of patients who had exophthalmos and whose hyperthyroidism, at the time of death, was in the stage in which basal metabolic rates are elevated and constitutional symptoms are present. As previously explained, the observations, which recently have been made in cases in which basal metabolic rates were low, probably are not correctly informative as to what takes place in exophthalmic goiter when the basal metabolic rate is high. Our experience is no greater than is that of others. Since the introduction of iodine as a routine in the treatment of exophthalmic goiter, before thyroidectomy, the number of deaths has fallen to near the vanishing point, and permission to examine orbits, in the few bodies that are available, seems to be exceedingly difficult to obtain. What information we have we owe to Dr. L. B. Wilson, who tells us that in examinations, which he made before 1917, of eight patients who had active exophthalmic goiter with exophthalmos, he found very little fat, very little edema, and very little venous congestion. The muscles were small and obviously degenerated, presenting an appearance exactly like that of the weak muscles of the thigh which were studied in the same cases. Dr. Wilson expressed the tentative opinion that the exophthalmos could be explained by the weakness and relaxation of the rectus muscles. This conforms to the view which previously has been expressed by Traube, von Recklinghausen, Dalrymple, and others, and it is supported by our finding of a positive correlation between weakness of the quadriceps extensor muscles and exophthalmos, in the cases that have been reviewed for this occasion.

SUMMARY

Forward displacement of the eyeball occurs in a variety of general diseases, but is unusual except in exophthalmic goiter. In syphilis, in tuberculosis, in neurofibromatosis, in multiple xanthomatosis, and in other conditions in which granulomatous masses or tumors may fill the orbit, the explanation of any resulting exophthalmos is obvious. In the hemorrhagic diseases, including rickets, scurvy, and the various forms of leukemia, intraorbital hemorrhage may produce exophthalmos which, under these circumstances, is perfectly understandable. In nephritis, hypertension, and other conditions of so-called sympathicotonia,

true proptosis is very unusual, contrary to opinions recorded in the literature, although retraction of the lids and possibly other factors, which contribute to a facial expression somewhat like that observed in exophthalmic goiter, frequently are encountered.

The experimental production of exophthalmos must be evaluated with due consideration to the danger of applying to man observations that are made on animals, which normally are immune to exophthalmic goiter. Clinical experience indicates that the immediate cause of exophthalmos in exophthalmic goiter is dependent on actual changes in the function of the thyroid gland.

In large groups of patients, the presence or absence of exophthalmos in exophthalmic goiter is correlated directly with the severity of the hyperthyroidism, as measured both by the basal metabolic rate and the presence of weakness of the quadriceps femoris muscles. In the period after 1930, during which the severity of exophthalmic goiter has been milder, the incidence of associated exophthalmos has diminished to 40 per cent. Its incidence before 1922 was almost 70 per cent.

In exophthalmic goiter, exophthalmos may develop or progress in two rather distinct phases of the disease: first, it may occur in association with an elevated basal metabolic rate and with the constitutional symptoms of the disease; it also may appear in an otherwise quiescent phase of the condition, when the basal metabolic rate is within, or even below, normal levels and when the constitutional symptoms are absent or, at least, attenuated. Exophthalmos which develops when the basal metabolic rate is elevated in exophthalmic goiter is usually moderate in degree. Edema of the lids is present in only a small proportion of the cases, and it is usually slight. The proptosis almost always is bilateral and is about equal, or nearly equal, in both eyes; it usually is associated with spasm of the muscles of the lids, and one or more of the characteristic signs, which have been described by Dalrymple, Stellwag, Graefe, Möbius, and W. H. Wilder. Weakness of the external ocular muscles, which is indicated by the occurrence of Möbius' sign, is common, but ophthalmoplegia and strabismus are unusual. Remission of the exophthalmos usually follows the decrease in the basal metabolic rate and remission of the other manifestations of the disease, which are brought about by thyroidectomy.

Exophthalmos which develops or progresses when the basal metabolic rates, in the course of exophthalmic goiter, are within,

or even below, normal levels, and when the constitutional symptoms are absent, is unusual. Zimmerman referred to it as "paradoxical exophthalmos." Its degree as a rule is much greater than is that of the exophthalmos which is associated with elevated basal metabolic rates. Edema of the lids and chemosis of the conjunctiva are the rule. They may be extreme, and may lead to corneal ulceration and panophthalmitis. The condition has been termed malignant exophthalmos. The exophthalmos in these cases is very likely to be unilateral, or if not unilateral, it is likely to affect the eyes unequally. Ophthalmoplegia is common; on the other hand, spasm of the muscles of the lids is not marked and, in consequence, the characteristic "stare" of exophthalmic goiter is little in evidence.

The exophthalmos, which occurs with the elevated basal metabolic rates of exophthalmic goiter, perhaps may be explained by spastic contraction of the orbital fibers of Müller's muscle, which acts against the weakened rectus muscles; the absence of mydriasis under these conditions may be accounted for by the observation of Labbé and his associates that thyroxin has not only a sympathomimetic action but that it also stimulates parasympathetic elements of the autonomic nervous system. We, unfortunately, possess no recent reports of examinations of the orbit in this condition. The information which is available indicates that there is little fat, little edema, and very little venous congestion, and that the extraocular muscles are small and degenerated.

The explanation for the mechanism of the exophthalmos which occurs in patients who have low basal metabolic rates is not apparent. Why should the external rectus muscles become swollen and hardened, and enlarge to several times their normal thickness? Why should edema develop in the lids when it was absent in the stage featured by a high basal metabolic rate? The evidence from the laboratory suggests that overfunction of the anterior lobe of the pituitary body may play a part in the production of this abnormality. Another possibility is presented by the two-product hypothesis of H. S. Plummer, which is based on the assumption that the abnormal product continues to act after the output of the normal product, thyroxin, has been curtailed. The edematous contents of the orbit are comparable in some respects to localized subcutaneous areas of mucinous edema, which are found in rare cases of exophthalmic goiter, particularly after thyroidectomy. Whatever the mechanism, it is diffi-

cult to understand why a few patients should develop this paradoxical exophthalmos after subtotal resection of the thyroid gland for exophthalmic goiter, when the large majority lose what exophthalmos they had before, and give no sign of its recurrence. Further investigation of this question is under consideration.

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SYMPOSIUM:

THE ETIOLOGY OF EXOPHTHALMOS

COMPLICATIONS; CAUSES FROM PRIMARY LESIONS IN THE ORBIT; SURGERY

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A SYMPOSIUM on a given subject is really analogous to taking stock. We attempt to determine just where we are at the present time in regard to the problem in hand. When all has been said, we are sometimes reminded of the light that Milton described in hell as being just bright enough to make the darkness visible.

However, thinking in retrospect over the particular phases of exophthalmos with which I am concerned, I find quite a few encouraging signs of progress. This manifests itself strikingly in regard to the histogenesis, histology and accurate terminology of tumors. The older reports on orbital tumors are in many instances without much value because of loose, inaccurate terms which fail to classify the new-growths properly in a manner that assures uniform understanding.* The use of many different terms to designate tumors with a similar histogenesis has thus led to confusion and has retarded our knowledge of the various groups. With the more accurate classification of these tumors, it is now possible to state with more certainty, in regard to the various groups, their usual clinical course, their clinical peculiarities, their prognosis, their sensitiveness to radiation and their usual reaction to surgery.

* Sarcomas are often reported as belonging to the small or large round-cell type, although now such a diagnosis is considered insufficient. An attempt is made to state the genesis of the tumor by designating the type of cell, such as myosarcoma, neuro-sarcoma, osteo-sarcoma, lympho-sarcoma, etc. Formerly, the mixed tumors of the lachrymal gland were called endotheliomata, and were, therefore, confused with the primary and secondary meningiomata. Likewise, the varied manifestations of the meningiomata were called all kinds of tumors.

This paper deals mainly with the primary tumors of the orbit and also with surgical measures employed in cases of exophthalmos. Before considering these topics, though, certain complications suffered by the eyeball will be considered. One of the most frequent complications of the eye resulting from exophthalmos is the exposure ulcer of the cornea. In testing whether or not the lids protect the cornea properly, it is important to have the patients passively close the lids as in sleep and not actively squeeze. The earliest manifestations of corneal exposure are irritation of the eye and punctate areas over the lower one-third of the cornea which stain with fluorescin. If unheeded, these are the forerunner of an ulcer which, if unarrested, can progress to perforation and the loss of the eye. The presence of pressure or inflammation around the optic nerve frequently causes papilloedema and, in due time, atrophy. The same factors may also cause a thrombosis of the central retinal vein. A tumor in the orbit may push the scleral wall in as well as push the eyeball out. In the early stages, this indentation of the sclera may occur before the eye is proptosed, and in such a case the hyperopia acquired is significant. If the indentation of the sclera is localized, it may in some ways resemble a flat detachment of the retina.

Acquired tissue around the globe, whether neoplastic or inflammatory in origin, may produce glaucoma presumably by embarrassing the vortex system of veins. An orbital tumor may not only indent the sclera but even cause glaucoma before producing an exophthalmos.

The presence of an orbital tumor in the majority of cases causes some amblyopia, and not infrequently amaurosis. Most observers have attributed it to pressure of the tumor mass on the optic nerve. This does not seem to be the only factor, though, for the optic nerve may be completely imbedded in a meningioma and the eye still have normal vision and fields. On the other hand, such soft, pliable tumors as the hemangiomata cause amblyopia or amaurosis in three-fourths of the cases, and some of these tumors are not located in the muscle funnel or near the optic nerve.

Stretching of the optic nerve occasioned by the exophthalmos does not seem to be a very important factor. Sometimes the vision is markedly reduced with only 3 to 4 mm. of proptosis, and, on the other hand, an enormous protrusion may

cause very little amblyopia. For example, in one instance, the length of the optic nerve was estimated from x-rays to be more than twice its normal length, and still this patient had 20/30 vision and normal fields. The optic nerve from the optic foramen to the globe has a course represented roughly like the letter "S" and it is this slack which allows the free excursion of the eyeball and its protrusion without undue stretching.

Hyperopia and hyperopic astigmatism, caused by the tumor pushing the sclera in, form another cause of impaired vision. Due to the irregularity of the astigmatism, it usually cannot be corrected with lenses. It seems probable that still another factor must exist to explain satisfactorily the reduced vision in some instances. This must be a toxin emanating from the growth which causes a dysfunction of the nerve.

Primary tumors of the orbit may be benign histologically, but locally malignant in the sense that their slow growth may impair the function of the eye or reach the brain. To this group belong the hemangioma, the glioma of the optic nerve, the meningioma of the optic nerve sheath, and the mixed tumor of the lachrymal gland, all of which grow very slowly over a period usually of years.

HEMANGIOMA

Hemangioma is the most common primary tumor of the orbit and is fortunately the most satisfactory with which to deal because it is usually encapsulated and is radium-sensitive. It is probably entirely congenital in origin, although it frequently grows so slowly that it gives no sign of its presence until the patient is in the first or second decade.

Two characteristics of some importance are the failure of these tumors to affect the motility of the eye, and the variations in the size of the tumor and thus the extent of the exophthalmos. These variations may be spontaneous, due to a variable stasis in the venous circulation, or they may be produced by any manœuvre which increases the congestion in the jugular area, such as compression of the vein, bending the head, crying, coughing, etc. The same conditions produce exophthalmos in the condition known as varix orbitæ or intermittent exophthalmos, and, therefore, some confusion may arise.

In the case of varix orbitæ, the exophthalmos is provoked only by the above mentioned conditions which congest the jug-

ular area, and disappears when the patient is in the ordinary standing, sitting, or lying positions. The condition usually manifests itself first in middle age and is due to varicosities of the orbital veins which probably occur as a result of an atresia of the foramen through which the jugular vein leaves the skull. It is apt to appear on the left side, for normally the foramen is smaller on this side.

MIXED TUMORS OF THE LACHRYMAL GLAND

The large majority of new-growths of the lachrymal gland contain tumor tissue, some of which is ectodermal and some mesodermal in origin. These growths are, therefore, called mixed tumors and probably constitute fully 95% of the neoplasms that arise from the lachrymal gland. Pure carcinoma of the gland is an extreme rarity. Sarcoma of the gland has never been reported.

These mixed tumors are congenital, although the average age at which they manifest themselves is forty years. Van Duyse reports a case in which, during the course of an operation for the removal of a portion of the lachrymal gland because of epiphora, he accidentally encountered a mixed tumor the size of a pea lying between the main gland and the accessory gland. This substantiates the belief that these tumors arise from isolated displaced and undifferentiated embryonic rests, and not from the developed gland tissue. Because these mixed tumors have the potentialities of forming so many types of tissue, they naturally vary a great deal in their component structures. The term cylindroma has been employed to designate a certain manifestation of this tumor in which the epithelial element predominates in cylinder formation with a myxomatous stroma. The mixed tumors are not benign, as Lane reports on ninety-five cases in which there was 12.5% mortality and 20% recurrence. They are not just locally malignant, for 7% showed distal metastasis. The growth becomes progressively malignant after each incomplete removal.

INTRA-NEURAL TUMORS

According to Verhoeff, all primary intra-neural tumors of the optic nerve are composed of neuroglia and, therefore, are correctly called glioma. Clinically, a glioma of the optic nerve is extremely slow in growing, and produces exophthalmos grad-

ually in children in the first decade. Vision is usually affected before the onset of exophthalmos. Verhoeff discusses very interestingly the mode of origin and growth of these tumors. Ordinarily a tumor elsewhere evidently arises from a small focus and as it grows it pushes aside and invades the surrounding tissue. In the case of glioma, whether it be in the optic nerve, brain or cord, the growth increases in size by causing the preexisting neuroglia in the vicinity of the growth to proliferate. Sometimes this glial proliferation is seen as far forward as the disc and contiguous retina, where in some instances it may reach considerable size and form a white, even a cystic, mass. Also, glial proliferation accompanying a glioma of the nerve may be noted as far back as the hypophysis. Verhoeff's explanation of the proliferation of the neuroglia of the disc and contiguous retina, and perhaps also of the hypophysis, is that some abnormal stimulating factor is supplied in increasing abundance by or from the actively proliferating neuroglia of the tumor. This would also explain the remarkable fact that there is not a single instance in the literature in which a recurrence of a glioma of the optic nerve has occurred, although in perhaps the majority of cases the removal had been incomplete.

EXTRA-NEURAL TUMORS

The extra-neural tumors of the optic nerve, or meningiomata, have the varied histology of the same tumor arising from the meninges of the brain and cord.

They are particularly prone to produce changes in the contiguous bone, which changes, together with the fact that they affect adults, serve to distinguish them clinically from the intraneural tumors. They encapsulate, but the capsule tends to adhere to surrounding structures, which, in the case of the extra-ocular muscles, often causes injury when the tumor is removed without the globe.

SARCOMA

Sarcoma of the orbit may arise from a great variety of cell types. It may be connective tissue, muscle, nerve, periosteum, fat or lymphocytes. An analysis of fourteen cases from the Head and Neck Clinic of the Memorial Hospital showed that every one of the twelve patients who could be traced lived only on an average of one year after the first manifestation of the new-growth. Regardless of the treatment, whether

it was radiation, surgery, or both, a rapidly fatal course occurred. These figures are no more discouraging, though, than those for sarcoma elsewhere.

It is usually said that the younger the patient the more malignant the tumor. This was true in our cases where the youngest patient was fifteen months old and the tumor proved fatal in five months. The average age of our cases was ten years.

PRIMARY CYSTS

Of the primary orbital cysts, one of the most interesting types is the congenital coloboma cyst or retinocele. The cyst may enlarge after birth and produce a very marked exophthalmos. It always arises from the under surface of the optic nerve at the site where the nerve enters the sclera, and is due embryologically to a faulty closure of the fetal cleft, which allows a bulging or diverticulum of the retinal layer. The cysts are, therefore, lined with neuroglia or rudimentary retina. There is usually an associated microphthalmos, but occasionally the eye is of normal size and appearance. In such instances the question of whether or not a new-growth is the cause of the exophthalmos may arise. The presence of an anomalous condition of the disc, iris, etc., is then important in making the correct diagnosis.

The dermoid cysts usually occupy the orbital margin in the region of the brow temporally, and less often nasally. Very rarely they appear deeper in the orbit or extend from the usual superficial location back along the bony wall to the retro-bulbar region. In extremely rare instances they may be hour-glass in shape, with one side in the orbit and the other side in the temporal fossa. In such instances, pressing the jaws firmly together or pressure on the external portion of the cyst will press the contents into the internal portion and produce a further protrusion of the globe.

Although these cysts are congenital, they may enlarge slowly or sometimes even grow so rapidly in the course of weeks or months as to produce pain.

PSEUDO-TUMORS

In some instances it is impossible to say from the clinical examination whether a real primary tumor or a pseudo-tumor exists in the orbit. That these two conditions can simulate each

other in every detail is evidenced by the fact that, of 30 cases of pseudo-tumors analyzed, 15, or 50%, had exenterations of the orbit for a supposed tumor which proved by histologic examination to be a chronic inflammation instead. For some reason these cases of pseudo-tumors are seldom mentioned in our literature, maybe because one hesitates to broadcast one's mistakes. Nevertheless, pseudo-tumors of the orbit are fairly common, and it is often a difficult problem to differentiate them from real tumors. In the group of cases of pseudo-tumors analyzed, the following characteristics were found:

1. The average exophthalmos produced was 7 mm.
2. A mass which may be firm, well demarcated and discreet, was palpable in the orbit in one-half of the cases.
3. The vision was affected in two-thirds of the cases and usually returned to normal after regression.
4. Complete regression, which often proceeds to enophthalmos and sinking of the tarso-orbital fascia, took place usually in four to five months.
5. The condition is never recurrent.
6. Occasionally there was prodromal transitory edema of the lid and conjunctiva some weeks or months preceding the appearance of the exophthalmos.

The most important points in differentiating a pseudo-tumor of the orbit from a real primary tumor are the following:

1. Primary tumors of the orbit occur mostly in the first decade of life and rarely after the second decade. The average age for pseudo-tumors is forty-five years.
2. The onset of the exophthalmos in primary tumors is insidious and gradual. In pseudo-tumors the onset is relatively sudden, occurring in several weeks in most instances.
3. Primary tumors of the orbit are never bilateral. Pseudo-tumors affect both eyes in one-third of the cases, but the second eye becomes involved four to nine months after the first.
4. Primary, as well as secondary, tumors of the orbit not infrequently show x-ray changes of the orbital bones, whereas pseudo-tumors characteristically do not.
5. Primary tumors usually cause no pain and no swelling of the conjunctiva or lids. Pseudo-tumors in one-half of the cases give rise to some pain and some edema around the eye.

The microscopic examination of tissue in these cases shows chronic inflammation with certain distinguishing characteristics. There may be fibroblastic repair in all stages from the denser hyaloid, almost anuclear tissue of long standing to the delicate fibroglia and richly nuclear tissue of more recent production. Foci of lymphocytes are scattered through this repair tissue, and occasionally real lymph follicles with germinal centers are encountered. Blood-vessels are plentiful, their intima often showing proliferation to such an extent that the lumen appears almost occluded, and their media and adventitia being thickened with sclerosis which has sometimes undergone hyaloid degeneration. When biopsies are taken from this tissue, there is often a tendency to bleeding, which, in one instance, necessitated packing and secondary closure. This tendency is explained by the vascular changes described.

The etiology of pseudo-tumors is not known. There is no evidence clinically, or histologically, that the lesion is tuberculous. Some observers claim that the lesion is due to a very late manifestation of syphilis which has run its course and, therefore, gives a negative Wassermann. Of the group studied, only two had a positive blood Wassermann and one a positive spinal fluid but a negative blood Wassermann. There are reports of regression of the exophthalmos under the usual treatment for late syphilis, but the effect may be absorptive rather than specific, and the condition when untreated subsides in the course of some months. The histologic characteristics of the lesion and in particular the vascular changes described above are not inconsistent with lues, but there is a preponderance of evidence which fails to substantiate a luetic etiology.

In connection with the pseudonoma, it may not be amiss to mention the fact that a high myopia may produce a prominence of the eye, which, if unilateral, can be misleading. I know of one such case of 30 D. which was investigated from all angles in an effort to explain a 4 mm. exophthalmos before the myopia was appreciated as the cause. Also, a paralysis of one or more of the rectus muscles or a tenotomy of a rectus muscle may produce an exophthalmos of 2-3 mm.

OPERATIONS

The operations indicated in cases of exophthalmos may be for cosmetic purposes, for more protection to the cornea, for the excision of a tumor, or for the relief of pain.

No. 1 — Cosmetic Purposes: Particularly in cases of exophthalmic goitre, where the protrusion of the eye is a definite blemish, the appearance can be materially improved by a shortening of the palpebral fissure at the external canthus. A technique which will give the normal acute angle to the junction of the upper and lower lids at the external canthus and not a rounded obtuse angle is desirable. Such a technique, which we find satisfactory, is the following one advocated by Wheeler (Fig. 1).

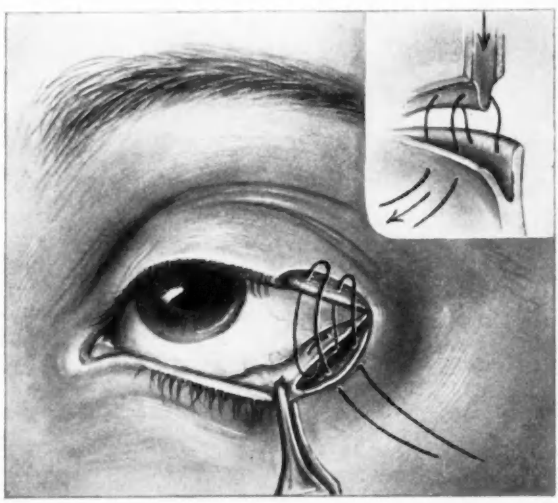

FIG. 1 — (Reese) — Wheeler Tarsorrhaphy. Over the desired distance of shortening, the conjunctiva and the skin, including the cilia along the margin of the upper lid, are excised. This leaves a denuded short tongue of tissue including the tarsus. Over the same corresponding distance along the lower lid, the cilia are excised and a slit is made between the skin and the conjunctiva into which the denuded tongue of tissue from the upper lid is pulled and sutured with a double-arm silk suture. The inset is a cross-section of the procedure.

Also, in cases of hyperthyroidism, where the retraction of the upper lid rather than the protrusion of the eye constitutes the greater blemish, a recession of the levator muscle, as advocated by Goldstein, may be indicated. In marked cases, when the exophthalmos and the retraction of the upper lid are both important factors, the fissure shortening may be combined with the recession of the levator.

No. 2 — For Protection of the Cornea: If the lids fail to protect the cornea, particularly when passively closed during sleep, it may be necessary to resort to surgery to prevent an exposure ulcer or to prevent progression and promote healing of an ulcer already present. Surgical measures which tend to forestall the development of an exposure ulcer of the cornea are the fissure shortening and the recession of the levator. However, if an ulcer is well established, it may be necessary to produce adhesions along the lid margins, thereby closing the palbebral fissure in order to secure proper protection of the cornea. If adhesions between the lid margins are produced properly, they will usually remain indefinitely. The course of the ulcer can be watched and atropine instilled through the spaces intervening between the adhesions. At any time the adhesion can be severed, leaving no deformity of the lid margins. Merely suturing the lids together with mattress sutures is not sufficient, as these will hold, usually, not more than four or five days.

In cases of malignant progressive exophthalmos, sometimes seen in hyperthyroidism, Naffziger has advocated a decompression of the orbit by resecting the orbital roof. As this is done from the anterior cranial fossa, it comes under the domain of the neurological surgeon.

No. 3 — For Removal of an Orbital Tumor: Tumors that are located temporal to the muscle funnel, or in the muscle funnel, can be very satisfactorily approached by a transconjunctival route in the following manner. A canthotomy is performed and the fibers of the external canthal ligament, together with the tarso-orbital fascia are divided (Fig. 2). The conjunctiva is incised along the fornix to the vertical meridian above and below the canthotomy. This conjunctiva is dissected free. If the tumor is in the muscle funnel, the external rectus muscle must be severed at its insertion (Fig. 3). By retracting the globe nasally, very good access to the orbit is obtained. We believe that any

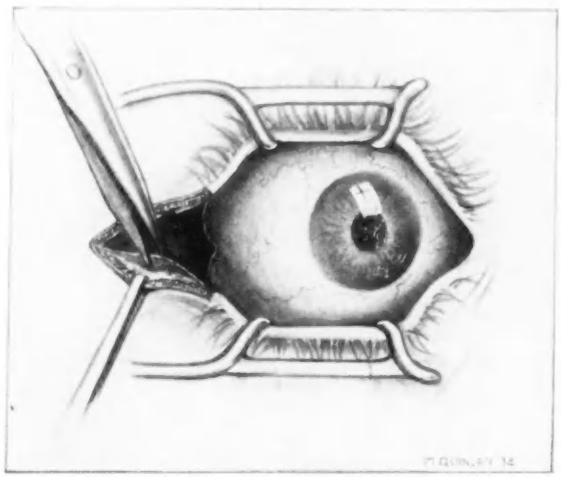

FIG. 2 — (Reese) — Transconjunctival Approach to Orbit. A canthotomy has been done and the external canthal ligament along with the tarso-orbital fascia is being incised.

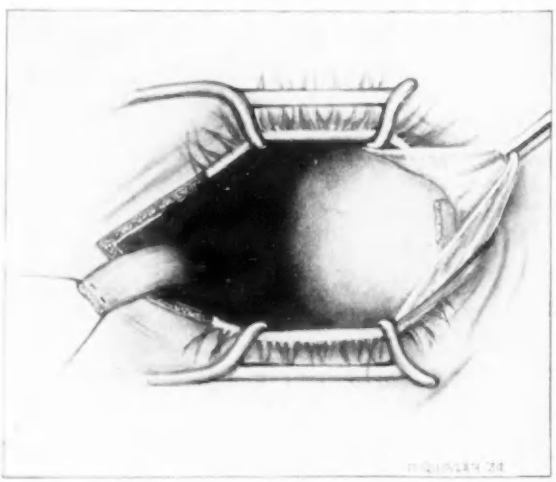

FIG. 3 — (Reese) — Transconjunctival Approach to Orbit. The conjunctiva has been incised along the fornix to the vertical meridian above and below the canthotomy and dissected free. The external rectus tendon has been severed at its insertion and reflected. The globe is retracted nasally.

tumor can be removed as satisfactorily in this manner as by the Krönlein method and that the Krönlein operation is seldom, if ever, necessary. If one needs slightly more exposure than is afforded by the above-described procedure, then some of the margin of the bony orbit temporally can be rongeured without causing any cosmetic blemish. This additional procedure is rarely necessary.

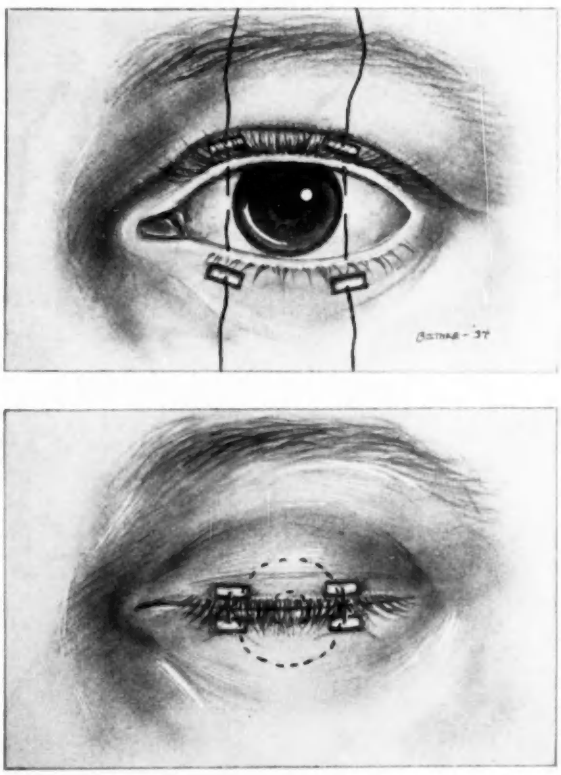

FIG. 4 — (Reese) — The upper drawing shows the placement of sutures for holding the globe in the primary position. The lower drawing shows the sutures tied over rubber pigs.

The temporal transconjunctival approach gives sufficient access to the orbit for the removal of tumors located anywhere but nasally. When tumors occur nasally in the orbit, neither this approach nor the Krönlein is satisfactory, because, if the muscle funnel must be crossed in approaching the tumor, then

damage to the extra-ocular muscles, to the posterior ciliary vessels and nerves, and to the optic nerve occurs to such an extent that it is not feasible to attempt to retain the globe. If the tumor is definitely in the nasal side of the orbit, the same trans-conjunctival approach can be utilized nasally. Even when tumors lie in the muscle funnel, and particularly in the apex of the orbit, and can be satisfactorily approached and removed, they sometimes are so adherent to the extra-ocular muscles that the muscles must be damaged in removing them. When such damage to the extra-ocular muscles is extensive, there will result an exophthalmos, a poorly aligned eye and ptosis. When one or several extra-ocular muscles are damaged, it is sometimes advisable to fix the eye in the primary position so that the repair tissue formed later in the orbit around the globe will fix it in good alignment and with no proptosis. This can be accomplished by passing a silk suture through the lid margins and through the episclera at the limbus nasally and temporally, and tying over the surface of the lids (Fig. 4). A tenotomy of the opposing muscle may be advisable.

The fate of a globe left in situ, when the optic nerve has been excised along with the orbital tumor, is interesting. Immediately after excision of the nerve from the globe a view of the fundus with the ophthalmoscope shows a slightly pale fundus; but the arteries, although somewhat narrower than normal, are filled with blood and the veins do not appear remarkable. In the course of some months, marked pigmentary changes occur over the fundus and even in the disc (Fig. 5 — colorplate). Retinitis proliferans is sometimes noted. An irido-cyclitis with secondary glaucoma may develop, and ultimately enucleation may be necessary. Verhoeff microscopically examined an eye of which the optic nerve had been severed several years previously. He found an extreme degree of atrophy of the choroid and retina with pigmentation of the latter by cells migrating from the proliferated pigment epithelium. As obstruction of the central vessels alone does not cause such changes, it is assumed that they occur as a result of the severing of the posterior ciliary arteries.

When the new-growth is extensive, or diffuse, or, if it cannot or should not be locally extirpated, then an exenteration is indicated. In such cases, it is, of course, advisable to remove all but the skin of the lids. The skin of the upper lid is inverted upon the roof of the orbit and likewise the skin of the lower

lid is utilized to cover the floor of the orbit. If the remainder of the orbit is allowed to granulate, discharge occurs over a long period of time, requiring frequent dressings, sometimes even for months. Therefore, at the time of the exenteration, if a Thiersch graft is placed in the orbit, it will take readily and leave a clean, non-discharging, odorless orbit one week after the operation. It is surprising how readily a graft takes on bare and supposedly deperiosteomized bone.

No. 4 — For the Relief of Pain: Sometimes patients with tumors in the orbit, which have extended there secondarily from the sinuses, nasopharynx, etc., will experience severe pain. Although the patient may be deemed inoperable and incurable, they can often be relieved from pain by an exenteration.

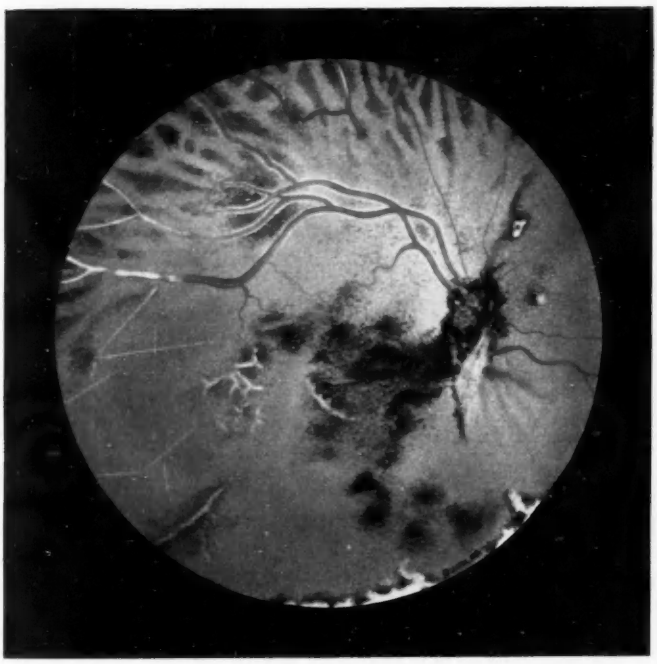

FIG. 5 — (Reese) — A meningioma arising from the optic nerve sheaths was excised leaving the globe in situ. Three months later this fundus drawing was made. (Patient of Dr. T. H. Johnson). There are diffuse pigment changes, particularly around and over the site of the disc. Also, in the region of the disc are two sites of retinitis proliferans. The blood vessels in places appear as white lines. In the periphery below are white atrophic areas.

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SYMPOSIUM:

THE ETIOLOGY OF EXOPHTHALMOS

LESIONS OF NASAL AND CRANIAL CAVITIES

EXOPHTHALMUS FROM SURGICAL DISEASES OF (A) THE CRANIAL CAVITY, (B) THE NASAL SINUSES, AND (C) THE BONY ORBIT, ESPECIALLY AS TO INVOLVEMENT OF THE PROTECTIVE RETRO-BULBAR SPACE

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NEWARK

THE BONY ORBIT is (a) a goblet-shaped cone, open in front, within which, posterior to the eyeball, there is (b) a closed fascio-muscular space[1,2] for which I would suggest the term, "protective retro-bulbar space," the surgical importance of which has not been appreciated. Overfilling of this space, even from slight circulatory alterations, which in other parts of the body gives rise to flushing and congestion, as in hyperthyroidism, here causes exophthalmus.

In suppurative and traumatic disturbances resulting from neighborhood lesions, this space is the most fundamental, although the least evident, in the production of exophthalmus; for any factor outside the peri-orbita requires (a) a marked constriction of the capacity of the bony goblet-shaped cone to produce proptosis, but (b) a slight overfilling of the protective retro-bulbar space, whether it come from the cranial cavity or from the orbital or nasal walls will occasion exophthalmus.

1. Wolff, Eugene, *The Anatomy of the Eye and Orbit*, H. K. Lewis & Co., Ltd., London, 1933, p. 310.
2. Zuckerkandl, E., *Atlas der topographischen Anatomie des Menschen*, Wien, Fig. 102, s. 107, 1904.

Again, suppurative insult to the nervous mechanism within the space produces pathognomonic fundus changes of the highest diagnostic and prognostic significance which, until now, have not been properly interpreted.

The Closed Protective Visual Sense Space Posterior to the Eye

The retro-bulbar space, within the goblet-shaped bony cone of the orbit, is a special visual sense protective space. Its function is the protection of the end organ — the eye — against injury from trauma, and from infection of the neighboring respiratory apparatus. It is a closed mesoblastic space which contains, however, ectodermal elements. First, it develops in conjunction with that part of the special visual sense bone forming the optic foramen, the compact tissue of which, in the embryo, grows outward from the spheno-ethmoidal cartilage attached to the special sense nasal capsule.[3] Again, within it is a specific type of ligamento-muscular sling, the annulus of Zinn (annulus tendinus communis), to which the ocular muscles are attached and that has a dural element in its composition.

This closed retro-bulbar space is filled with fat (a) in an almost liquid state. In addition, it has other unique anatomical features, among which are that (b) there are no lymphatics in it, and (c) its veins are without valves.

Surgical Anatomy of the Non-lymphatic Protective Retro-bulbar Space

The space is limited in front by (a) the septum orbitale and (b) Tenon's capsule; posteriorly by (c) the annulus of Zinn, and, laterally by (d) the fascio-membranous muscular cone of the orbital muscles.

The septum orbitale, or palpebral fascia, which forms the broad ligaments of the eyelids, is a floating membrane firmly attached to the bony rim of the orbit. It forms a barrier to the passage of extravasations from the orbit to the lids.

Tenon's capsule — the fascia bulb — is also a more or less "floating membrane"; it acts as an additional barrier, largely between the globe and other contents of the orbit.[4] Both mem-

3. Fawcett, Prof., *Notes on the Development of the Human Sphenoid*, Jour. Anat. and Physiol., 44:208-222, 1909-10.
4. Whitnall, S. E., *Anatomy of the Human Orbit*, Henry Frowde and Hodder & Stoughton, London, 1921, p. 291.

branes can withstand considerable pressure. Injections (Charpy[5]) into the retro-bulbar space ranging from 40 to 50 mm. Hg., i.e., double the normal tension of the eye, are prevented from passing into the eyelids (Heerfordt[6]).

Lying almost loose in the lobulated semi-liquid fat in the retro-bulbar space are the arteries, veins and nervous connections — the optic nerve and the ciliary ganglia, and its afferent and efferent communications — for the eye.

The Valveless Venous Plexus Within the Space

The orbital venous system is in reality a plexus, a network of small vessels.

The upper plexus usually unites to form the chief vein, the superior ophthalmic vein, which communicates with the angular vein in front. The superior ophthalmic vein becomes constricted at its entrance into the cavernous sinus. As with all venous mesh-works, its connections are extremely intricate, as well as the point of exit of the superior ophthalmic, which may perforate the annulus of Zinn, or leave the orbit below, above, or external to it. The vena centrales retinal, as a rule, leaves the optic nerve about 10 mm. from the globe, and opens directly into the cavernous sinus; but may end in the superior ophthalmic vein, to which it invariably sends a good-sized anastemostic branch (Sesemann).

The lower venous system of the orbit originates in the orbital fat beneath the globe, as a still finer network, and may continue as such throughout its entire course, communicating freely with the superior ophthalmic, or may unite to form an interior ophthalmic vein. As a rule, it enters the cavernous sinus separate from the superior ophthalmic below the annulus of Zinn (Freund), but it may discharge all of its blood into the superior system.

Veins from the inferior ophthalmic plexus pass through the inferior orbital fissure communicating with the plexi in the infratemporal fossa and pterygo-maxillary spaces (Henle), where they, in turn, again communicate with veins from the

5. Charpy, *Structure topographique des paupières*, Bibliographie Anatomique, xxl. 2, p. 65, 1911.
6. Heerfordt, *Über das Emphysema der Orbita*, Graefe's Archiv für Ophthalmologie, 58:123, 1904.

upper and lower jaws and with the intricate plexus surrounding the condyle.[7]

Anomalies of the Veins of the Retro-orbital Space of Surgical Importance

(a) The inferior ophthalmic may be the chief vein and may anastemose with the angular. (b) The superior occasionally passes directly into the Sylvian vein (middle cerebral) without entering the cavernous, in which case a phlebitic meningitis may develop from an orbital infection without the cavernous sinus being affected.

(c) At times, the ethmoid veins empty directly into the cavernous sinus, in which event a suppurative ethmoiditis may occasion a cavernous sinus thrombosis — a very infrequent origin of the intracranial lesion — from a suppuration outside of the peri-orbita, without involvement of the retro-bulbar space.

Routes of Infection into the Protective Retro-bulbar Space

Thus, anatomically, the retro-bulbar space can become infected by an endophlebitis coming (a) from in front, by way of the superior ophthalmic plexus; (b) from below, from the pterygoid plexus or through the inferior ophthalmic plexus; or (c) mesially from the nose. The endophlebitis of the vessels within the space may remain (a) a thrombophlebitis of one or other of the ophthalmic veins (Case 2 — Child W.); or it may (b) form a local abscess within the cone; although retro-bulbar space infection usually is but a part of (c) a generalized orbital phlegmon.

A suppurative lesion of the space usually occasions (1) a cavernous sinusitis, with (2) a terminal meningitis. However, infection of the space itself may arise secondarily from a cavernous sinus phlebitis (Case 4 — Man E., and Case 5 — Man G.).

Clinical Evidences of the Protective Properties that the Orbital Tissues Possess for the Control and the Prevention of Spreading Infections

Clinical facts of the greatest surgical significance are that: (a) enucleation of the eyeball never results in an actively spreading phlegmon, no matter how septically the operation may

7. Poirier, P., and Charpy, A., *Traité d'Anatomie Humaine*, Masson et Cie., Paris, 1903, pp. 932 and 946.

have been performed; and, that (b) transplants of fat buried within the orbit invariably heal kindly, although the graft cannot be absolutely aseptic. Furthermore, (c) both of the above occur without lymphatics, which, in other subdermal tissues containing fat, is the chief protective mechanism in the elimination of infection.

The Protective Forces Within the Orbit Against an Infection of the Retro-bulbar Space

Anatomically, passive protection against infection from the face is furnished by (a) the delicate but effective fascia orbito-palpebralis and Tenon's capsule; but, I believe, that it is (b) the active locally phagocytic elements furnished by the reticulum of the retro-bulbar space, and (c) the digestive action of the semi-liquid fat[8] contained within the reticulum, both within and external to the space, that are the chief factors in this immunity.

Clinical Evidence that Widening of the Retro-bulbar Space by a Surgical Decompression Relieves Exophthalmus

In reviewing the literature, one is struck by the number of cases in which an exploration by a Kroenlein osteoplastic flap for a supposed tumor, diagnosed as such because of the presence of exophthalmus, and although no pathology was found, the operation resulted in the return of the eye to its normal position (Königstein[9]).

This can only be accounted for on the basis of a decompression of the bulbar space, as recession does not occur after multiple punctures around the eye.

A Specific Type of Retro-bulbar Space Infection from the Ordinary Orbital Phlegmon

The specific type of infection, to which I would call attention, are those cases of orbital phlegmon, almost invariably associated with a cavernous sinus phlebitis, in which (a) the veins are the mode of transmission of the infection; and (b) the vessels within the fascio-muscular retro-bulbar cone are involved.

8. Hartwell, S. W., *Surgical Wounds in Human Beings; Histologic Study of Healing with Practical Applications; Fibrous Healing*, Arch. Surg., 2:76-96, (July) 1930.
9. Abstracted in Am. Ency. Ophth., VII, p. 4855, Cleveland Press, Chicago, Ill., 1915.

The Ordinary Orbital Abscess Resulting from Nasal Suppuration Not of Phlebitic Origin

Usually, an orbital abscess resulting from an ethmoidal sinus suppuration is (a) localized tissue suppuration, situated (b) external to the peri-orbita. Neither the veins nor the retro-bulbar space are involved, although the latter may be the seat of a reactive œdema, thus, materially augmenting the exophthalmus.

Discovery of the Unique Character of the Ophthalmoscopic Picture from Infection Confined to the Retro-bulbar Space and its Disappearance After an Orbital Decompression

In re-reading my histories of cavernous sinus infection, of which I now have seen thirty-eight cases, I was struck by the frequency with which (a) the vision of the affected eye had suddenly been impaired; examination of the fundus showing (b) whiteness of the nerve, (c) obliteration of the retinal artery, (d) the veins being either dilated or not involved. Associated with these symptoms, there are several records of (e) inequality of the pupillary reactions, and (f) an early steaminess of the cornea; the latter being out of proportion to the slight degree of the exophthalmus that was present.

At the time, I had attributed the fundus change to the lodgment of emboli of a clot in the arterial supply to the eye from the cavernous sinus. However, the ophthalmoscopic picture was not that of an embolic occlusion of the central retinal artery, in that, while the disc was whitish, the retinal tissue itself was not œdematous, and there was no mention in any of my notes of the presence of a cherry-colored ring around the macula; nor was it the picture of a thrombosis of the retinal vessels, because of the absence of hemorrhages in the fundus.

In one case of cavernous sinus phlebitis, the sudden blindness (Case 5 — Man G.) (with whitish nerve head and constricted arteries) was the first really alarming symptom, the patient having been working but a few hours previously. The exophthalmus at the time of the onset of blindness was very slight. At his death, the following day, examination of the orbital contents disclosed a localized area of purulent inflammatory infiltration which was confined within the fascio-muscular cone of the apex (Case 5 — Man G.).

Again, in a case of extradural abscess which was situated above the optic nerve and which followed a fracture through the optic plate of the frontal bone, there was a whiteness of the nerve head with constricted arteries the day after the fracture. The preoperative diagnosis (Dr. John V. Bissett) was "hemorrhage into the optic nerve sheath," a condition which I had previously described,[10,11] and for which I have operated with a moderate degree of success in several cases. At the operation no hemorrhage into the sheath was found, but the decompression of the orbit by the removal of depressed bone and the evacuation of hemorrhagic clots, was immediately followed by return of the circulation to the fundus with the restoration of vision (Case 1 — Girl S.).

Whiteness of Nerve Head from Depressed Fracture of the Orbit Relieved by Elevation of the Fracture

Case 1 — Girl S. (Dr. John V. Bissett, Newark City Hospital) — A young girl was struck by an automobile and suffered a compound depressed fracture of the frontal bone involving both orbits. The fracture extended across the nose. The entire orbital roof was loose. The following day the optic nerves were white. Hemorrhage in the optic sheath was thought to be the cause of the whiteness of the nerve of the right side.

Operation: Brain elevated over the orbital plate. Fracture seen extending posteriorly over the orbital plate, apparently, entering the orbital foramen.

The day following the operation the whiteness of the nerve had disappeared. The child did well for several days, then developed pneumonia, a stiff neck, sudden coma and died. The cerebro-spinal fluid contained streptococci.

The autopsy showed a fracture extending through the entire orbital plate of the right side, across the cribiform plate and over the orbital roof and of the great wing of the sphenoid of the left side.

The orbital plate of the right side was easily loosened. There was extradural hemorrhage with an extradural abscess of this region. There was no hemorrhage into the sheath. "There is a marked constriction where the optic nerve goes through the optic foramen. It is difficult to dissect the nerves away from the bone." The post-mortem

10. Eagleton, Wells P., *Fracture of the Skull, Importance of Early Diagnosis and Operative Treatment*, Arch. Surg., 3:140-153, (July) 1921.
11. Eagleton, Wells P., *Brain Abscess*, p. 169, Macmillan Co., New York, 1922.

revealed greatly increased intracranial pressure; however, during life, there had been no papilloedema or injection of the veins at the fundus. The whiteness of the nerve was thought by Dr. Harrison Martland to be due to an "arterial coma" from a paralyzing influence on the sympathetic, which caused obliteration of the arteries.

Phlebitis Confined Within the Retro-bulbar Space Is Surgically Analogous to Ludwig's Angina

Surgically, isolated retro-bulbar space infection simulates Ludwig's angina, as it is an acutely extending infection situated within unyielding tissue. In the case of Ludwig's angina, the infection is confined between two fascial planes, the weaker yielding to swelling, causes elevation of the tongue. In infection of the retro-bulbar space, posterio-lateral expansion is prevented by the bone of the orbit containing the musculo-membranous cone and the annulus of Zinn. The inflammatory œdema must move the eye forward, carrying with it Tenon's capsule.

In infection of the retro-bulbar space, as in Ludwig's angina in the early stage, there is no pus, the tissues being infiltrated with serum. However, pressure and the virulence of infection rapidly causes extensive sloughing by necrosis of tissues (Case 5 — Man G.), prior to which the nervous mechanism controlling the intraocular circulation reacts uniquely, because of the visibility of the retinal vessels with the aid of the ophthalmoscope.

Stupeur Artérielle

The sudden septic insult acting upon the exposed ciliary ganglia, and possibly upon the vaso-vasorum surrounding the ophthalmic artery, causes a protective reaction in the artery occasioning a *stupeur artérielle*, which, in turn, gives rise to (a) sudden diminution or loss of vision, associated with (b) whiteness of the nerve head, and (c) contraction of the arteries of the retina, its veins being lightly affected.

Rapid Formation of Abscess in Space

A stage of serous effusion within the space cannot long continue as the valveless veins, having no adventitious coat, break down with the formation of an abscess, the usual method by which Nature localizes vascular infections in all well-protected areas.

Rapid Orbital Necrosis Without Abscess Formation from a Phlebitic Infection Largely Confined to the Retro-bulbar Space, Associated with Fulminating Purulent Cavernous Sinus from a Stye on the Eyelid

CASE 2.—CHILD W. (Drs. Henry C. Barkhorn and John V. Bissett).—A boy, five days previously, had a stye on his right eye which opened spontaneously and had disappeared on November 29th. On Sunday, December 6th, he had a right-sided earache. However, the drum examination was negative. The following day, the right lid became swollen. On the next day (December 8th), there was a right proptosis.

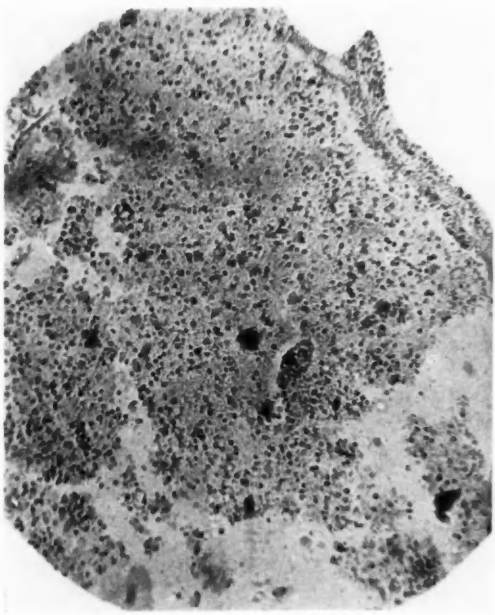

FIG. 1.—(Eagleton).—(Case 2.—Boy W.).—Suppurative Thrombophlebitis of Ophthalmic Vein. Showing colonies of bacteria. *(Low power).*

EXAMINATION: Now showed a greatly swollen lid of the right eye, the tissue around being swollen and œdematous. There was considerable proptosis and slight chemosis. The ocular conjunctiva contained, in its outer corner, a large number of veins running parallel with the long axis of the orbit. The lids of the left eye also were slightly swollen and there was slight chemosis.

EXAMINATION OF THE FUNDI: (a) The right disc was absolutely white. There were (b) no arteries to be seen. The veins, however, (c) were full. There was (d) a slight haziness of the cornea. The left fundus presented a similar condition, but not as marked; that is, white nerves, small arteries and congested veins.

Rapid Terminal Cerebral Œdema — During the day the child had six convulsions. After the first convulsion his temperature reached 105° F. and it remained high until his death.

FIG. 2 — (Eagleton) — (Case 2 — Boy W.) — Suppurative Thrombophlebitis of Ophthalmic Vein. Showing one colony of bacteria. *(High power).*

Leucocytosis 19,000. The blood culture showed staphylococcus aureus. (Smear and culture from right eye showed staphylococcus aureus).

He died the same day — three days after the beginning of acute symptoms, and ten days after the original stye infection.

POST-MORTEM

BASE OF SKULL: *General Œdema Around Veins* — There is general distension of all the veins of the base. The pituitary region is œdematous. The olfactory nerves are

œdematous. The ophthalmic nerve on the right side is more œdematous than the left. There are small hemorrhages in the dura of the right middle fossa region. The right cavernous sinus is œdematous — more so than the left.

Pus in Anterior Extensions of Both Caverns—On opening the right cavernous sinus, its anterior extension contains pus.

On the left side, the anterior extension of the cavernous contains more pus than the right side. Running along the floor of the middle fossa there is what looks like an

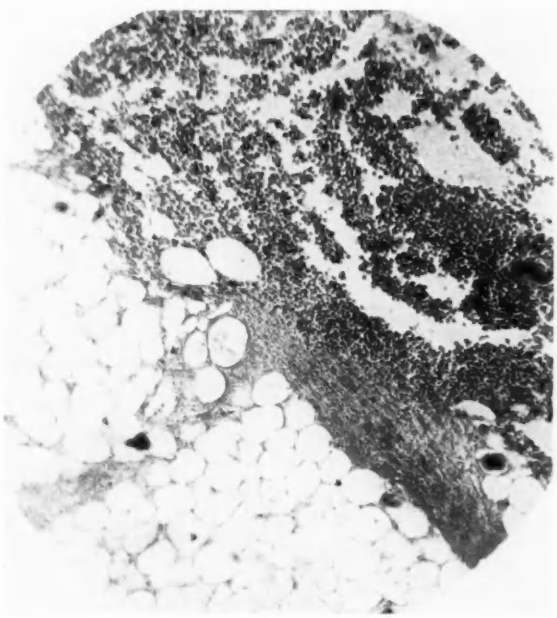

FIG. 3—(Eagleton)—(Case 2—Boy W.)—Suppurative Thrombophlebitis of Ophthalmic Vein. Orbital Tissue (right). Extensive Microscopical Suppuration. *(Low power).*

anterior cavernous sinus which has opened and contains a clot with many bands in it, the same as are seen in the cavernous sinus. The carotid artery of the left side is more œdematous than the right. The floor of the left middle fossa shows pus under the dura.

COMMENT: "The disease coming from in front, apparently, extended a little further backward into the superior petrosal of the left than it did in the right side."

Purulent Thrombophlebitis of Ophthalmic Veins:

ORBITS: The right orbit, opened from behind, macroscopically, showed no cellulitis, œdema or free pus, but there was *grumous pus in the ophthalmic veins.* Smear from the right orbit showed staphylococcus aureus. The left orbit showed the same condition, excepting that the ophthalmic vein was smaller than the right. Smear from the left orbit also showed staphylococcus aureus.

MICROSCOPICAL: Section of the ophthalmic veins — "suppurative thrombophlebitis" (see Figs. 1 and 2). The orbital fat of the right side presented the picture of microscopical suppuration (see Fig. 3). The orbital fat of the left side showed microscopical necrosis.

Rapid Development of Whiteness of Fundus in a Case of Fulminating Cavernous Sinus Phlebitis and Meningitis from Pimple on Nose and Nasal Sinusitis

CASE 3 — BOY R. — A young boy (seen in consultation with Dr. J. Reiner). On November 8th, he had a soreness on the tip of his nose, with a cold in his head. On the following day he vomited and had a headache.

Two days later he complained of a severe throbbing headache and was nauseated, and he had a temperature of 101° F. with a rapid pulse. There were no chills but he felt chilly. On November 15th (one week after the pimple on his nose) there was a slight redness of the upper lid of the left eye and his severe headache returned. His temperature rapidly rose to 104° F.

On the following day (November 16th) the left eye was completely closed, with redness and swelling of the upper lid and there was a slight exophthalmus. The vision rapidly failed.

Examination of the left fundus showed the disc to be paler than the right. The veins of both fundi were full, but those of the right disc were very full and tortuous. *During the day vessels of the left fundus grew progressively paler.*

X-ray showed cloudiness of the left frontal sinus, ethmoids and antrum.

DIAGNOSIS: Beginning suppurative meningitis from fulminating cavernous sinus phlebitis and pan-sinusitis.

OPERATION: Primary ligated the common carotid with fascia lata. Evisceration of the left orbit; incision and drainage of the cavernous sinus, which bled slightly. Culture from the pus from the frontal sinus showed streptococcus and micrococcus catarrhalis.

On admission to the hospital, the boy complained of double vision; he had a stiff neck and a Kernig. The lumbar puncture showed 1700 cells with reduction of glucose. He rapidly developed a general meningitis.

Death followed meningitis and venous sepsis with a terminal infarct into his lung. There was a loud systolic murmur over the heart.

Fundus Signs of Arterial Strangulation in One Eye and Venous Obstruction in the Opposite from a Fulminating Cavernous Phlebitis and Meningitis

Case 4 — Man E. — A young man, ten days before examination, had a pimple on the left side of his nose. His nose was not sore, but he had chills. He thought he had "a cold." On the second day (Sunday), he was quite ill; temperature was around 101° F. The swelling of the nose increased, extending to the lid of the left eye. On Friday, the swelling passed to the lid of the opposite eye. His temperature rose to 104° and 105° F. and he had a very rapid pulse. That night (Friday), one week following the onset of the illness, he became delirious.

At the time of my examination, the following Monday, he was semi-comatose, moaning constantly, had a stiff neck, and the facial cyanosis of profound toxemia. He was manifestly dying of a terminal meningitis.

Examination showed a bilateral exophthalmus which was much more marked in the right eye — the one secondarily involved. The veins in the lids were dilated and tortuous. There was a *bluish black chemosis of the right conjunctiva*. The eyeball was fixed. *Fundus of the right eye* showed œdema around the nerve. The veins on this disc were blackened, but *no arteries were present*. The picture was that of strangulation of the arterial system.

Left Eye: The left lids were œdematous with moderately dilated veins. There was a slight chemosis of the left conjunctiva. The pupil was small. The fundus veins were black and very prominent; the picture of venous obstruction.

Impression: "It would seem from the history that the infection passed from the left facial vein into the left ophthalmic vein causing obstruction, but not a complete blockage of the venous return. It then rapidly extended from the left orbit into the cavernous sinus and entered the right orbit, where it is now causing death of tissue."

The Surgical Importance of Recognition of the Phlebitic Character of the Orbital Phlegmon

In phlebitic orbital phlegmon, there is a peculiar blueness of the vessels of the conjunctiva, many of which run parallel with the globe (Case 2 — Boy W.). In such a case, the usual exploration for a pocket of pus by multiple punctures outside or inside (Butler[12]) of the muscular cone, or after the division of one or both tarsal ligaments (Lawson and Tweely[13]), in my experience, has but too frequently converted a semi-quiescent phlebitic infection into an actively advancing one.

Sudden Blindness with Whiteness of the Fundus from a Retrograde Invasion of the Retro-bulbar Space Followed by a Fulminating Cerebral Sepsis,[14] the Result of a Subacute Cavernous Sinus Thrombophlebitis with but Slight Symptoms from a Stye of the Eyelid

CASE 5 — MAN G. — A middle aged man had a stye on his right eyelid. Four days later (April 28th), the muscles of his neck felt stiff and he had some difficulty in swallowing, but he continued to work. While the stye on the right lid was "doing well," the left lid became slightly swollen and sore.

On the following day (April 29th), although his temperature was 101° F., he felt well and again returned to work.

On April 30th, he had violent pain in the left side of his head. He was seen by his physician (Dr. E. S. Goudy, of Harrison, N. J.) at 5 o'clock in the afternoon, at which time the patient made no complaint of the eye, and the swelling of the lid was so slight as to escape attention. Two hours later (7:00 p.m.), he suddenly lost the sight of the left eye, and the pain in his head progressively became worse. By (8:30 p.m.) the time of my examination, there was a bilateral exophthalmus — slight swelling of the right eye, but marked of the left; slight chemosis in the lower part of the conjunctiva and a beginning haziness of the left cornea.

FUNDI: The left fundus showed general whiteness, "as if the blood supply was almost absent." However, beyond

12. Butler, T. Harrison, Discussion on *The Diagnostic Significance of Proptosis*, Trans. Ophthal. Soc. of the United Kingdom, 1923.
13. Lawson, George, *Plastic Cellulitis of the Orbit*, Trans. Ophthal. Soc. of the United Kingdom, 15:186, 1895.
14. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case 11, p. 29), The Macmillan Co., New York, 1926.

the disc one vein was markedly dilated. The right fundus was normal.

Death, in coma, followed twelve hours after the onset of blindness, and seven days after the primary stye infection.

POST-MORTEM: There was pus in the anterior end of the left cavernous sinus, which contained a clot, extending from the left cavernous sinus through the posterior and anterior circular sinuses into the right cavernous sinus. There was a slight hemorrhage about the left carotid artery and left anterior clinoid process.

ORBITS: The left orbit was œdematous with hemorrhagic infiltration of the muscles and of the retro-bulbar tissues behind the eye. The same condition was present on the right side but it was less marked.

Sphenoid sinuses — small blood clot — no pus.

BRAIN: The meninges showed no signs of inflammation, but there was moderate dilatation of the veins of the pia and the cortex of the brain was flattened from increased intracranial pressure.

Differentiation Between Exophthalmus Resulting from Erysipelas and a Phlebitic Orbital Phlegmon

In the early stages of the disease, I frequently have been unable to distinguish between an exophthalmus resulting from an erysipelas in the early stages and a phlebitic orbital phlegmon. Today, I am inclined to regard any proptosis as significant that the infection is deeper than the skin in which the erysipelas streptococcus travels. It is my further belief that those cases of proptosis that subsequently develop into erysipelas are primarily phlebitic phlegmons in which the streptococcus causing the phlegmon has been transmuted into erysipelas; for it is now recognized that streptococci from a scarlet fever erythematosis of the skin may be transmuted with a resulting phlegmonous cellulitis, or even an osteomyelitis.

Relief of Exophthalmus in the Homolateral Eye and Restoration of Vision Following Blindness with White Disc and Obliteration of Retinal Arteries in the Contralateral Eye from Retrobulbar Phlegmon by Ligation of the Carotid Artery. Complications of a Purulent Cavernous Phlebitis from Extensive Phlegmonous Gangrene of the Head, the Result of a Carbuncle of the Nose

Case 6 — Man D. (Seen with Dr. S. A. Cosgrove) — A young man, on November 25th, had a "pimple" on the left side of his nose, which was incised. On the following day, he worked but felt rather sick. He again returned to work the next day (November 27th) during which time he had a chill and his face began to swell. The chill recurred. Two days later, exophthalmus of the left eye appeared.

Examination: (December 1st) He was conscious and answered questions intelligibly, but his temperature was 106° F. There was a "gangrenous carbuncle of the nose" which had points of gangrene and hemorrhagic areas in it. Little areas of gangrene had extended over on his face, and red lines ran into the scalp, which was the seat of an œdematous infiltration, involving both sides of the neck.

The left globe was bulging extremely. The pupil was small. The eye was blind.

Left Fundus: The disc of the left fundus was almost absolutely white, with only one vessel above and one below it. I could not tell whether they were veins or arteries.

His right eye was also proptosed and he had a chemosis, but not so marked as in the left. At this time the *right fundus* did not show any indistinctness of the margin of the disc, but the vessels were small, the veins were not overlarge. *Vision of right eye normal* as he could see objects well.

Rapid onset of blindness and fundus changes as the right retro-bulbar space was involved. However, three hours later, he was totally blind in his right eye, as well as in his left, the right fundus now gave the same appearance as the left had three hours previously.

Operation: On making an incision in the neck, all its tissues were found juicy. Ligation of the left external and internal carotid arteries, just above the bifurcation, with ligation of the first ascending branch of the external carotid. This had an immediate effect on the exophthalmus of the left eye, which receded perceptibly.

Left orbit eviscerated and cavernous sinus drained.

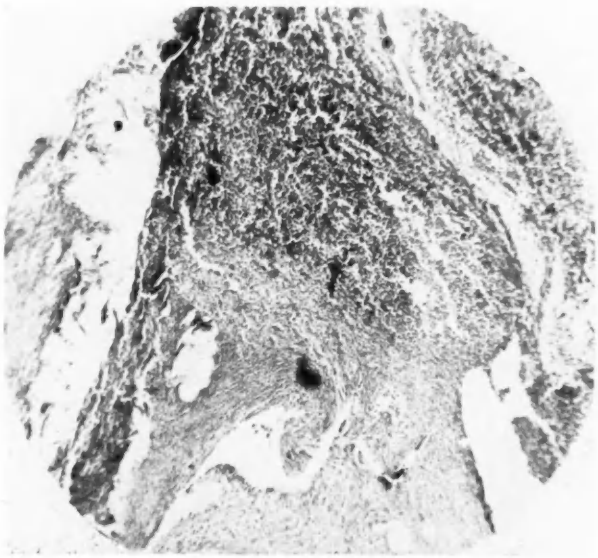

FIG. 4—(Eagleton)—(Case 6—Man D.)—Suppuration in Retro-orbital Tissue. Pyogenic Retro-orbital Infection showing cellular infiltration.

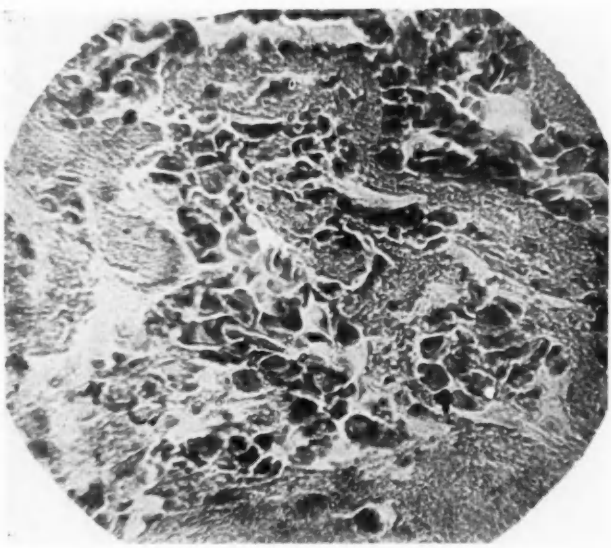

FIG. 5—(Eagleton)—(Case 6—Man D.)—Suppuration in Retro-orbital Tissue. Pyogenic Retro-orbital Infection. *(High power).*

Restoration of Vision of Right Eye (opposite to carotid ligation). Following the operation, the man was able to count fingers promptly with his right eye.

SUBSEQUENT HISTORY: Symptoms of pneumonia rapidly appeared; pain in the side of his chest, expectorated bloody, sticky sputum: Blood count 7000 with 85% polynuclears.

He died of cardiac failure on the following day; seven days after the beginning of the "pimple" in his nose, five days after the first chill, and three days after the appearance of exophthalmus. His death was undoubtedly due to pneumonic involvement by septic infarcts, and not from meningitis from the cavernous sinus, as there was no stiff neck or Kernig, and he was conscious to the end.

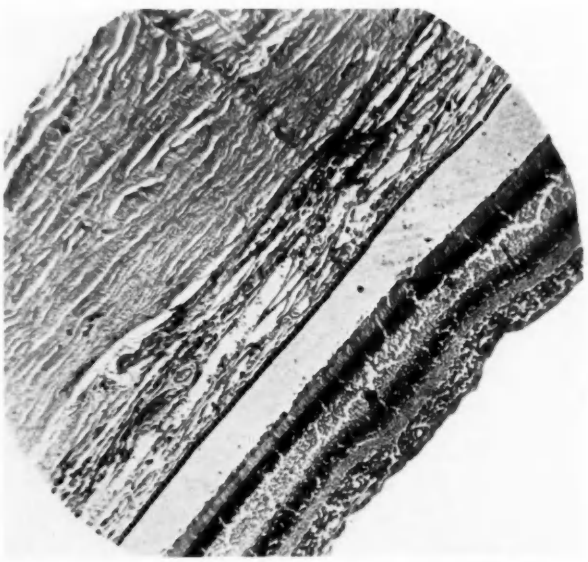

FIG. 6—(Eagleton)—(Case 6.—Man D.)—Suppuration in Retro-orbital Tissue. Pyogenic Retro-orbital Infection. Eye Free.

POST-MORTEM: *Base of Skull* — The whole median area of the middle fossa had multiple small hemorrhages in the dura, and hemorrhage into the pituitary. The vein that runs forward along the floor of the left middle fossa was enormously engorged and raised while the corresponding vein on the right side appeared flat and empty. The lateral veins of the left middle fossa were much larger than those of the right side.

The left cavernous sinus was fuller than the right. Its cavity was filled with *grumous clot, but no pus.* The outer end of the left superior petrosal was free, there being a distinct line of demarcation where it entered the cavernous. A vein running along the small wing of the sphenoid was very large and thrombosed.

The right cavernous sinus also *contained a grumous clot.* The right carotid artery in the cavernous was thickened and softened.

ORBITAL EXAMINATION: Tissues of the right orbit were juicy (see Figs. 4 and 5). The right ophthalmic vein was, apparently, thrombosed. The left ophthalmic vein also contained a clot. The enucleated eye was free of disease (see Fig. 6).

ACCESSORY SINUSES: The sphenoid contains bloody secretion.

BRAIN: The brain was whitish from pressure.

The Ganglionic Origin of Whiteness of Nerve Head and Obliteration of the Arteries in Infections of the Retro-bulbar Space

It has long been known that the immediate effect of a traumatism to the brain is accompanied by a whiteness of the disc. This is followed on the next day by a congestion of all the vessels. The whiteness of the disc is (probably) due to "spasm of the arteries" from the trauma to the central mechanism of the sympathetic around the third ventricle of the brain. The arterial spasm has been named "*arterial coma*," or "*stupeur artérielle*" (Leriche).

Acute bacterial insult from a venous infection within the intra-membranous muscular cone, well posterior to the eyeball, apparently, occasions a similar arterial spasm, for I believe it is demonstrated from the post-mortems of Cases 6, 2 and 5 (Man D., Boy W., and Man G.) that the associated blindness and whiteness of the nerve of the contracted retinal arteries, while apparently of arterial origin, were primarily due to a venous infection within the retro-bulbar space situated so far posterior to the eyeball that the venous circulation from the retina was not affected. Certainly the arterial symptoms appeared coincident with a sudden septic thrombophlebitis of the retro-orbital veins.

An anatomical explanation is that the fundus picture is primarily due to the insult to the exposed nervous mechanism contained within the ciliary ganglia, the nervous mechanism reacting by contracting the ophthalmic artery.

Pupillary Changes from Ciliary Ganglion Involvement in Infections of the Retro-bulbar Space

That the disturbance is due primarily to the nervous system and not to the vascular system is shown by the effect on the pupil, as in Cases 13, 4 and 7 (Girl D., Man E., and Boy M.). The pupil was small on the side of the irritation of the ciliary ganglia in Cases 4 and 7 (Man E. and Boy M.), although it reacted consensually following ligation of the common carotid, which it did not do before, and in both instances the eye could be opened after ligation.

The ophthalmoscopic whiteness is in accordance with the fundus picture seen in the sudden blindness following the trauma from operations on the nose (Würdemann[15]) and following injections of paraffin.

The arterial spasm is, undoubtedly, a protective reaction. Consequently, I believe that in cases of infection of the retro-bulbar space, prompt drainage should relieve the spasm and restore the vision, as in Case 6 (Man D.), in which the sight returned by ligation of the opposite carotid artery, although the lesion in the orbit must have continued; again, as in Case 1 (Child S.) in which the whiteness of the fundus disappeared after the relief of pressure, the result of a fracture.

Exophthalmus with Blindness and Retinal Vascular Obliteration Caused by Nasal Operation with Subsequent Contraction of the Pupil

> Case 7 — Boy M. — A boy, seen in consultation with Drs. L. Peer and Lee Hughes, had a chronic bilateral sinusitis. The opening, by a competent man, of the right posterior ethmoidal cells, the bone of which was abnormally hard, was immediately followed by protrusion of the right eye. On coming out of ether, he was blind.
>
> On the following day, there was swelling of the lids with ecchymosis, a marked exophthalmus, a slight chemosis,

15. Würdemann, H. V., *Injuries of the Eye*, Cleveland Press, Chicago, Ill., 1912, pp. 205-6.

a complete paralysis of the third nerve, with ptosis and a widely dilated pupil. He had no perception of light. At first the fundus had been normal, but during the examination it gave the picture of a vascular occlusion; the arteries were very small, and were "kinked" over the disc. The veins, also, were very small. He was now able to recognize hand movements.

DIAGNOSIS: Fracture running through the orbit and optic foramen.

OPERATION: Under local anæsthesia, the right common carotid was ligated with fascia lata. *On tightening the ligature, there immediately was some recession of the eye and he was able to elevate his lids.*

Ether was then administered and a modified Kroenlein was performed, going as far back as possible in the removal of bone, followed by a vertical incision through one of the muscles. The orbital tissue was not under great tension. The optic nerve was freed. The passage of a hook under the optic nerve liberated a few clots of old blood. The optic nerve was not swollen. A small incision was made in it.

The operation had no effect on the vision or the third nerve paralysis, but the exophthalmus immediately disappeared.

SUBSEQUENT PECULIAR PUPILLARY REACTION — Several months later the right pupil was found to be smaller than the left. "It would seem as if the small pupil must be due to an injury of the ciliary ganglia — certainly not to the injury to the sympathetic in his neck." Fundus showed an area of blackish pigment in the macular region.

The early haziness of the cornea is also probably due to ciliary ganglia involvement in Case 2 (Child W.). In Case 13 (Girl D.), there was not only an interstitial keratitis, but the next day she had a low intraocular pressure.

COMMENT:

Hemorrhage Into the Conjunctiva — In Case 13, the hemorrhages into the conjunctiva were probably embolic infarcts, as she had albumin in the urine. In my experience, hemorrhages do occur in the retina in cases of cavernous sinus thrombophlebitis.

Fixation of Eyeball — The fixation of the eye muscles is probably due to the inflammatory exudate (Case 4 — Man E.).

Cavernous Sinus Thrombosis Resulting from Aural Suppuration, While Usually of the Slow Compensatory Thrombotic Type, May Present Arterial Ophthalmic Symptoms When an Acute Infection Enters the Retro-bulbar Space

The acute orbital phlebitis causing exophthalmus is usually part of a phlegmon from a phlebitic infection of the nasal orifice, the eyelids and the lip. In the fulminating cases, it may be associated with an extensive streptococcic erysipeloid infection of the skin (Case 6 — Man D.). The acute phlebitic cases invariably terminate in a cavernous sinus phlebitis, complicated by an *almost simultaneous meningitis.*[16]

This type of diffuse phlebitic phlegmon must be surgically recognized as a different pathological lesion from the slowly progressive low-grade compensatory thrombus due to infection of the middle ear, which enters the cavernous mesh from behind by way of the petrosal sinuses. The former is a diffuse endophlebitis of the small vessels, the latter a more or less localized abscess surrounded by a limiting clot within a large venous radicle. Either lesion, however, may cause *"stupeur artérielle"* by the sudden entrance of the infection into the protective retrobulbar space.[17]

Exophthalmus in Infections of the Posterior Part of the Cavernous Sinus the Result of Aural Suppuration

In the cases of compensatory thrombosis of the cavernous sinus originating from a suppurating ear, the exophthalmus is usually slight in degree and is apt to be of transient duration; because (a) the collateral venous return from the orbit is soon reëstablished, and (b) the venous stasis, which is the chief cause of the exophthalmus, rapidly subsides.[18]

I summarize, herein, five cases of subacute compensatory thrombosis venous infection from aural suppuration, three of which (a) retrograded through a petrosal sinus, and involved

16. Eagleton, Wells P., *Suppurative Meningitis of Otitic and Nasal Origin,* Arch. Otolaryngol., 15:885, (June) 1932.
17. Kalb reports a case of white disc from an aseptic thrombosis, as the patient had no rise in temperature during three months of illness. (*Ein Fall von Hirnsius Thrombose,* Berlin Klin. Wchnschr., p. 663, 1876).
18. Eagleton, Wells P., *The Carotid Venous Plexus as the Path of Infection in Thrombophlebitis of the Cavernous Sinus,* Arch. Surg., 15:275, (Aug.) 1927.

the cavernous; one (b) attacking the cavernous sinus directly from a diffuse suppuration of the bone marrow within the petrous apex, and one (c) originating from both cavernous and sphenoidal suppuration. The exophthalmus and chemosis of all were characterized by being (a) slight in degree, and (b) of transient duration. All were (c) unaccompanied by any change in the *arterial* supply of the retina.

> Case 8 — Girl W.[19] — Sometime after an operation for a mastoiditis, the child's eye appeared to be "dark blue,"[20] and she complained of pain in the eye. Suddenly, she became maniacal, and developed external rectus paralysis with other symptoms of petro-apiceal infection.
>
> A carotid ligation on the side of the mastoiditis and the evacuation of a granulomatous mass from the apex permanently cured her.

In this case, I am of the opinion that an acute venous congestion was the cause of the transient blueness around the eye, as I have seen all symptoms of apex suppuration promptly subside after a carotid ligation alone.[21]

> Case 9 — Boy G.[22] (Patient of Dr. E. A. Curtiss) — A young boy had an infection of the jugular bulb which extended into the cavernous sinus and occasioned a suppuration in the petrous apex and an abscess around the carotid artery. During the course of the pyæmia, he had two attacks of transient exophthalmus with but little or no chemosis. Both subsided within a few hours. The proptosis first appeared on October 2nd, at which time the cavernous sinus probably was attacked, and recurred on October 7th, when the sinus was completely obliterated.

He died from septic meningitis on November 3rd without exophthalmus or other evidence of venous congestion. The microscopical section showed an organized blood clot within the cavernous sinus, demonstrating the reparative process in a large venous trunk that may occur in one month's time.

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19. Eagleton, Wells P., *Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex (Case 4)*, Arch. Otolaryng., 13:386-422, (March) 1931.
20. Alexander, S., *Otogenic Thrombophlebitis of the Cavernous Sinus*, Monatsschrift f. Ohrenheilk. u. Laryngol., 59, H. 9, s. 971, 1925.
21. Vail, H. H., *Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex*, Trans. Amer. Otological Soc., Inc. pp. 117-119, 1932.
22. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case 24, p. 148), The Macmillan Co., New York, 1926.

CASE 10 — P.[23] — An elderly woman had so few symptoms from the infection in her ear that she was thought to have pneumonia; when, in reality, she was suffering from a chronic streptococcus mucosus (pneumococcus type III) sinus thrombophlebitis. The suppurative process gradually extended into the jugular bulb, then retrograded through the inferior petrosal into both cavernous sinuses.

The day before she became unconscious, from a terminal meningitis, there appeared a slight exophthalmus and chemosis, although the post-mortem showed that blocking of both cavernous sinuses had existed for some time.

Here, the obstruction of the venous return from the orbit took place so slowly that all signs of venous congestion were absent until the terminal meningitis augmented the general stasis by further interfering with the venous return from the anterior part of the head.

CASE 11 — WOMAN C. — A middle-aged woman was taken sick on November 16th with a "cold in her head and chest." She had buzzing in her left ear, with but little pain and no discharge. She became delirious; this was relieved by a spontaneous aural discharge. Following a mastoid operation, she left the hospital as cured.

On January 9th, one month following the operation, she came to the clinic of the Newark Eye and Ear Infirmary with a slight proptosis of her left eye. The next day the exophthalmus had disappeared. Subsequently, an operative trauma caused a recrudescence of a diffuse, but a latent osteomyelitic phlegmon within the substance of the marrow of the petrous apex, associated, as it is apt to be, with a suppurative meningitis.

At autopsy, both cavernous sinuses were found filled with pus, although there had been absence of proptosis and of all other evidence of venous stasis from the time of the invasion of the sinus, several weeks prior.

CASE 12 — MAN S.[24] — A man suffered from an acute infective cavernous sinus thrombosis complicating an otitis media and a sphenoidal sinus suppuration. During the third week of his illness, and two days prior to his death from general sepsis, a slight ocular œdema appeared.

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23. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis,* (Case 13, p. 38), The Macmillan Co., New York, 1926.

24. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis,* (Case 20, pp. 98-101), The Macmillan Co., New York, 1926.

Exophthalmus Intermittent

A few cases have been reported. In Calombo's[25] case, the exophthalmus was associated with a suppurating otitis and recurred with each successive otitis; but as the ocular protrusion could also be produced by pressure over the right jugular, or by turning the head to the right, and as it persisted after the aural suppuration had subsided, the intermittent proptosis was doubtless due, not to infection, but to a congenital anomaly of the vessels returning the venous flow from the orbit.

Temporary Exophthalmus and Chemosis from Continued Pressure on the Opposite Jugular During an Operation for Sinus Thrombosis, the Result of Aural Suppuration

I have seen two cases of œdema of the conjunctiva resulting from the pressure of a sandbag during an operation; one was accompanied by a bilateral exophthalmus. Both patients suffered from thrombosis of the veins emptying into the jugular bulb from aural suppuration.

Diagnostic Importance of an Induced Exophthalmus or Chemosis

Consequently, if continued pressure from a sandbag applied to the neck causes an ocular œdema, it is presumptive evidence that the infection in a venous sinus within the domain of the ear has extended into the cavernous sinus; otherwise, the return circulation from the eye could not be disturbed. Pressure chemosis and exophthalmus may be regarded as an expansion of Crowe's sign. In cases of thrombophlebitis of the venous sinus around the ear, it is diagnostic of involvement of the cavernous sinus.

Spontaneous Hemorrhages in the Opposite Conjunctiva in a Case of Otitic Thrombophlebitis, Exophthalmus and Chemosis from Continued Pressure on the Neck, Later Keratitis, Possibly from Sympathetic Nerve Involvement in the Orbital Apex and Low Intraocular Pressure

CASE 13 — H. D. — A young girl had pneumonia eight weeks previously. On the 8th day of the present illness, there was pain in the right ear, necessitating first a para-

25. Calombo, Ann. di ott., vol. 42, p. 602; abstracted in Am. Ency. Ophth., VII, p. 4860, Cleveland Press, Chicago, Ill., 1915.

centesis and later a mastoidectomy. This was followed by five weeks of aural sepsis, with an up and down temperature and irregular chills. She never had any headache.

At the time of my examination, there were small punctate hemorrhages into the conjunctiva of the opposite eye, and, although both pupils were small, the left (the contralateral) was smaller than the right.

OPERATION: Pus in the lateral sinus. No bleeding from the region of the jugular bulb.

While on the operating table, as the result of pressure of a sandbag, there gradually developed a bilateral exophthalmus with chemosis on the side of the free jugular. All exophthalmus and chemosis rapidly disappeared on its removal.

The influence of the Intraocular Tension and Keratitis from Venous Infection, Possibly with Ciliary Ganglionic Involvement — Later, it was found that the intraocular tension of both eyes was reduced and a keratitis appeared, which latter might have resulted from involvement of the ciliary ganglion. Slight indistinctness of the opposite fundus and an opposite external rectus paralysis accompanied the terminal basilar meningitis.

COMMENT: Hemorrhages into the left conjunctiva may have been due to emboli from her heart, as she had albumin in the urine.

CASE 14 — MAN R. W.[26] — A young man had pyæmia from an ascending septic thrombophlebitis originating from a septic sore throat with cervical adenitis. A purulent venous clot invaded the internal jugular vein, retrograded into the jugular bulb, the lateral sinus, and the petrosals, finally reaching the cavernous sinus.

During the whole course of the disease, the only symptom of venous obstruction of the ocular region was a chemosis of the conjunctiva from the pressure of a sandbag while the patient lay on the operating table at the time of the evacuation of a suppurating clot from the lateral sinus.

Differential Diagnosis Between Retro-bulbar Space Infection and the Usual Phlebitic Orbital Phlegmon

The vision of an exophthalmosed eye should be systematically tested at least once daily, as the patient is generally not aware that the sight has diminished, because the swelling of the lids engages all of his attention. Usually the loss of vision is

26. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case 17, p. 66), The Macmillan Co., New York, 1926.

only appreciated when the œdematous lids are forcibly opened, although, one of my patients (Case 5), suddenly announced, "I cannot see," which he discovered because he had but little swelling of the lid.

Treatment of Retro-bulbar Space Infections: Suggested Early Orbital Decompression and Incision for the Prevention of Cavernous Sinus Thrombophlebitis

As I have pathologically demonstrated (Case 2 — Child W.), (1) that (a) the obliteration of the arteries, and (b) the steaminess of the cornea, which occurs suddenly in cavernous sinus phlebitis, is not due to emboli, but to a septic inflammation affecting the arterial system within the fascial muscular retro-bulbar cone of the apex, and as (2) orbital decompression relieves exophthalmus, as demonstrated by the partial removal of the bony exostosis of the outer and upper orbital walls, I would lay down the dictum that in all phlebitic affections of the orbit, an orbital decompression should be performed immediately on the appearance of (a) whiteness of the disc, or (b) haziness of the cornea, no matter how slight the exophthalmus may be. The orbital decompression should be accompanied by an incision through the musculo-membranous layer of the retro-bulbar tissue, which allows of inspection and drainage.

The relief of a localized orbital phlegmon within the cone should suffice to control the infection in a considerable number of cases and, thus, prevents its extension into the cavernous sinus.

Technique of Latero-orbito-cranial Approach to the Retro-bulbar Space[27]

The solid bony angle of the temporo-frontal region and of the lachrymal fossa of the orbit is removed; thus, entering both the outer and superior area of the orbit through the lachrymal fossa and the anterior fossa of the skull. The orbito-cranial opening is extended posterio-mesially toward the optic foramen, going over the roof of the orbit, parallel with the orbital plate of the lesser wing of the sphenoid. The extensive removal of bone is followed by separation of the dura and its elevation from the floor of the anterior fossa.

27. Eagleton, Wells P., *A Case Showing the Possibility of Exploring the Orbital Contents from Behind Through the Cranial Fossa*, Arch. Ophth., 41:162, 1912.

An incision through the intra-muscular membrane now allows of exposure of the retro-bulbar contents of the orbit. By this method alone can a proper inspection of the retro-bulbar fascio-musculature cone be obtained and the space effectively drained without sacrificing the eye.

Operative Treatment of Purulent Thrombophlebitis of the Cavernous Sinus

Cavernous sinus thrombosis (a) from a phlebitic orbital phlegmon, or (b) from aural suppuration are true cases of venous sinus thrombophlebitis; i.e., with an abscess in the midst of a protecting thrombus.

The surgical indication, as in all venous sinus abscesses, is drainage, although even with pus in the cavernous sinus, the reactive endothelial elements within the vessel may first localize the infection by the formation of a clot both above and below the area of infection, and then causing phagocytosis of the causative bacteria. Thus, a low-grade compensatory septic process may be cured spontaneously (Case 9 — Boy G.).

Evacuation of a septic clot and drainage of the cavernous sinus can be accomplished by (1) evisceration of the orbit, and (2) the removal of the outer edge of the superior orbital fissure which exposes the cleft between the cavernous sinus and the dura of the middle fossa.[28] The cavernous sinus is easily located by passing a probe through either the superior or the inferior ophthalmic veins into the sinus, in which it is continued to its termination over the petrous apex. Incision of the vein and cavernous on the probe allows the insertion of a drain into the sinus.[29]

Carotid Ligation Prior to an Operation in the Cavernous Sinus

In all cases of cavernous sinus thrombosis the common carotid artery should primarily be ligated, but always under local anæsthesia; for if the ligation disturbs the circulation of the retina or causes cerebral anemia, the ligation must be but fractional.[30]

28. Mosher, H. P., *The Orbital Approach to the Cavernous Sinus,* Laryngoscope, 24:709, 1924.
29. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis,* (Case 22, pp. 136-137), The Macmillan Co., New York, 1926.
30. Eagleton, Wells P., *Localizing Value of the Ophthalmic Examination in Suppurative Disease of the Brain,* (Case 5), J. A. M. A., 92:715, (March) 1929.

Restoration of Vision Following Ligation of the Carotid

Carotid ligation in itself not only (a) places an inflamed cavernous sinus at rest, but it (b) relieves the venous stasis (see Fig. 7), which always accompanies infection,[31] as I have witnessed (1) the chemosis and the exophthalmus markedly subside immediately after carotid ligation (Case 6 — Man D.).

In addition, it certainly has (2) a marked influence on the ocular circulation, as in one case an eye which was totally blind had useful vision (Case 6 — Man D.) on the morning following the ligation.

FIG. 7 — (Eagleton) — Brain showing the influence of carotid ligation in the control of venous stasis on the left side of illustration.

Protection of the Eye After All Operations for Septic Lesions of the Orbit

All orbital operations, especially in septic lesions, should be completed by —

31. Eagleton, Wells P., Discussion of Dr. Vail's paper, (Case 2), Tr. Am. Otol. Soc., 22:119, 1932.

(1) Sewing the lids together, and
(2) Covering the eyelids with a large sheet of rubber tissue greased with vaseline.

This should *invariably* be done after any operation for nasal sinus suppuration, in which the septum orbitale is injured, as I have been called to three cases in which the sight of an eye had been destroyed by exophthalmus following a frontal sinus operation; the proptosis developed under the bandage, the pressure of which, with its dryness, caused the cornea to ulcerate.

Arterio-venous Communications Within the Cranium as the Cause of Exophthalmus

FREQUENCY OF PULSATING EXOPHTHALMUS OF INTRACRANIAL ORIGIN

Frost,[32] in 19 autopsies of pulsating exophthalmus, found:

Arterio-venous communication in	8
Affections of cavernous sinus in	2
Aneurysm of orbital portion of ophthalmic artery in	2
Orbital aneurysm in	3
Total verified cases as to cause	15
Undetermined	4
Total	19

that is, 10 out of 15, or 66%, of all verified cases of pulsating exophthalmus had some type of intracranial venous disturbance (Rivington[32]).

CAUSE AND SYMPTOMS

The pulsating is caused by the distended veins carrying arterial blood (Sattler[33]). The eye is apt to resemble an orbital phlegmon (see Fig. 8). The bruit may only be heard while the communication between the artery and sinus is small. When the tear widens, which may happen at any time, the bruit is apt to disappear.[34]

32. In Am. Encyc. Oph., 7:4861, Cleveland Press, Chicago, 1915.
33. In Am. Encyc. Oph., 7:4861, Cleveland Press, Chicago, 1915.
34. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case H, p. 130), The Macmillan Co., New York, 1926.

FIG. 8 — (Eagleton) — Traumatic arterio-venous communication resembling an orbital phlegmon.

Ausculatory Sounds Over the Orbit

The normal ausculatory sounds heard over the head or orbit have never been adequately described. To my ear, they are made up of two elements: (1) *Sounds from muscular contractions.* These are uneven in degree and irregularly present, but are always to be heard several times during the examination. (2) *Vascular sounds* which suggest, to my ear, (a) the confused noises of a number of operating machines as heard on entering a factory, augmented by (b) a rhythmical pulsation, like the throb of a large engine operating in the midst of the many irregularly revolving machines. This peculiar "rhythmic throb" is synchronous with the heart beat. Pressure over the homolateral carotid will often elucidate it, when it is otherwise not audible, as is the rule in cases of arterio-sclerosis.

I have frequently heard it over the site of a cerebral tumor, which speaks for the highly vascular nature of the growth (see Case 17). The bruit of arterio-venous communications is a blowing noise substituted for this pulsating sound.

Surgical Treatment of Pulsating Exophthalmus

In *pulsating exophthalmus* the surgical procedure must be dictated by the position of the lesion. If there is an arterio-venous aneurysm from a tear in the internal carotid in its course through the cavernous sinus, to attempt its cure by a *distal* ligation of the superior ophthalmic, but invites disaster.

The flow of arterial blood into an intracranial arterio-venous aneurysm may be stopped by ligation of the carotid, but it may be necessary to ligate the opposite carotid after a long interval.[35]

If, however, the exophthalmus is due to dilated veins in the orbit itself, a Kroenlein, or better still, the fronto-cerebral route will allow the application of a Cushing's silver clip to the dilated vessel.[36]

After treatment, the reduction of the brawny chemosis that remains for a long time after a protracted exophthalmus can be facilitated by systematic massage applied to the protruding conjunctiva, using a rubber glove covered with vaseline.

Differential Diagnosis of Arterial Aneurysm from Orbital Aneurysm

Horton[37] has demonstrated that it is possible to differentiate an intracranial arterio-venous communication from an orbital aneurysm by the blood from the jugular vein on the side of the arterial venous communication being of a darker color, and having a greater oxygen capacity (Van Slyke gasometric method) than the blood from the opposite jugular, while in the orbital aneurysms the blood from both jugulars is the same.

Exophthalmus from Fractures Through the Optic Foramen

In traumatic lesions, if an exophthalmus is present, it is due to the fracture passing through the annulus of Zinn, as hemorrhage into the orbit alone cannot cause it. If, however, the attachment of the external ocular muscles is injured, then hemorrhages may force the eye outward.

35. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case 1, p. 131), The Macmillan Co., New York, 1926.
36. Eagleton, Wells P., *Cavernous Sinus Thrombophlebitis*, (Case E, pp. 76-7), The Macmillan Co., New York, 1926.
37. Horton, B. T., *Intracranial Arterio-venous Fistula; Diagnosis by Discovery of Arterial Blood in Jugular Veins*, Proceedings of Staff Meetings of the Mayo Clinic, 5:178, (June 25) 1930.

Osteogenesis of Tumors of the Orbital Walls

The kind of primary tumor that may affect the orbital wall is dependent upon the generic origin of the bone.

(1) The outer orbital wall develops from cartilage, the resulting bone becoming filled with red marrow. For this reason, it can be the seat of (a) a simple hyperplasia — "a true bone callus" (see Case 15) — as the marrow bone proliferates easily, or (b) it may be the site of an osteochrondromatosis of all grades, ranging from benignity (Case 17) to the most active form of osteoblastic osteogenic sarcoma.[38]

(2) On the other hand, the inner wall of the orbit is a special sense-protective pneumatic bone. At birth, the bone spaces of the nasal capsule are filled with infantile fat marrow. During pneumatization the fat is absorbed, the pneumatized bone growth resulting in lamellar plates of compact tissue without bone marrow. Thus, in the bone of the inner orbital wall, which covers a nasal sinus, stimulation through chronic irritation from infection produces a type of (a) circumscribed ivory exostosis which is apt to be round when it occurs in the frontal sinus, or (b) lobulated and pedunculated when it develops within an ethmoidal cavity (see Case 16).

(3) *The roof of the orbit* again is made up of another type of special (visual) sense compact tissue bone. As far as I am aware, there is no record of the occurrence of a *primary bone* growth from the orbital roof.

Diffuse Bone Callus or Non-circumscribed Osteomas of the External Wall of the Orbit

In my experience, a diffuse osteoma, i.e., a diffuse bone callus, is apt to develop in the outer wall of the orbit following a slight trauma with a linear fracture of the bone (Case 15); this because the irritation of trauma causes the red bone marrow which fills the bone of the temporal fossa, including the outer orbital wall, to proliferate rapidly, forming bone callus; that is, vascular, periosteal and endosteal bone.

38. Geschickter, C. F., and Copeland, M. M., *Tumors of Bone*, published by American Journal of Cancer, New York, 1931, p. 2.

Exophthalmus from Diffuse Callus Formation of the Outer Wall Relieved by Orbital Decompression

CASE 15 — MRS. S. — A middle-aged woman had a goiter some years previously. On June 28th, 1925, she suffered an indirect injury to her head from which she became unconscious for a few seconds. Patient, from the time of the accident, complained of headache on the left side of her head and inability to concentrate.

EXAMINATION (March 19, 1926): Shows a localized swelling in the left temporal bone, just external to the angular process. It is slightly tender to pressure and is covered by enlarged veins. Marked exophthalmus of the left eye, which projects upward as well as outward.

Roentgen films revealed an area of density in the frontoparietal region, situated behind and above the orbit (Fig. 9).

FIG. 9 — (Eagleton) — Roentgenogram showing bony callus of outer orbital wall causing exophthalmus. Before operation.

OPERATION: The orbital wall and the skull was ¾ to 1 inch in thickness. Puncture of the dura allowed the escape of a large quantity of fluid where the growing bone was irritating the arachnoid.

POST-OPERATIVE DIAGNOSIS: A bone callus from trauma associated with an aseptic meningitis, the reaction of irritation from growing bone.

The operation was followed by relief of the exophthalmus and of all headache, although the x-rays still showed considerable thickening of the orbital region (Fig. 10).

December, 1933 (8 years later), the patient has continued well.

FIG. 10 — (Eagleton) — After operation (Case 15).

Figures 9 and 10 show that an orbital decompression with the removal of comparatively little of the bony contraction of the orbit will entirely relieve the exophthalmus. This is accomplished by allowing room for the lateral expansion, thus, freeing, as it were, the retro-bulbar space.

Ivory Exostosis of the Nasal Sinuses as the Cause of Exophthalmus

On the other hand, the inner wall formed by the bone of the nasal accessory sinus is composed of respirato-olfactory sense compact bone, and when irritated by infection and augmented by other unknown factors, may produce a new growth, a type of growth similar to normal compact bone of this region; i.e., a compact tissue exostosis.

Two Types of Osteoma

There are two types of osteoma which cause exophthalmus; viz.,

(1) The circumscribed round osteoma of the frontal sinus, and
(2) The pedunculated ethmo-orbital exostosis.

Both spring from a nasal sinus; the former from the frontal sinus and is apt to early extend into the cranial cavity, while the latter, as a rule, first invades the orbit.

Infections[39] *as the Cause of Exostosis Within the Accessory Nasal Sinuses*

My experience has been that these growths are always accompanied by an infection of the mucous membrane of the sinus from which the exostosis springs, for in all my cases, even when free pus was not seen at operation, — and it usually was present — a culture from the thickened mucous membrane revealed microörganisms. Consequently, I am of the opinion that an ivory exostosis of the nasal sinuses is the result of a chronic bacterial irritation, which irritation is applied to an area of osteogenic bone still capable of growing. Such areas are where, during embryogenesis, the cartilaginous bone of the nasal capsule is covered by an investing membranous bone from the face, the resulting bone being "appositional" or membranous bone, which is of the compact tissue variety. It is in the cleft between the cartilaginous and membranous bone that pneumatization occurs. Such areas, at birth, contain fat marrow; they grow actively during the first two decades of life. Thus, under the

39. Cushing is of the opinion that the ethmo-orbital exostoses are due to an unsuspected fracture at the junction of the orbital plate with the ethmoid capsule, stimulating into activity a cartilaginous anlage of bone growth. (*Cranial Changes Associated with Meningioma Dural Endothelioma*, Surg. Gyn. & Obstetrics, 48:231, 1929).

stimulation of infection within a sinus an ivory exostosis may develop, taking its lobulated shape from the ethmoid cells. A pedunculated osteoma of the ethmoid is apt to extend early into the orbit, causing marked exophthalmus, long before it invades the cranial cavity.

As the growth increases, the part protruding beyond the sinus from which it springs is surrounded by a "collar" of normal nasal sinus compact tissue bone.

Surgical Removal of the Collar

The surgical removal of an osteoma or exostosis of a nasal sinus is easily accomplished if the wedge-shaped "collar" of normal bone is removed by a powerful electrically-driven brazier. As soon as the outer border of growth is free, the entire growth can be "rocked" out of its bed with ease.

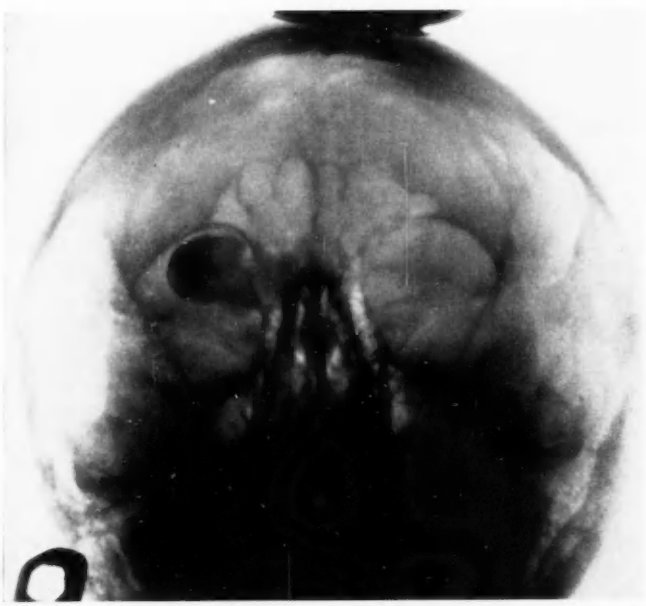

FIG. 11 — (Eagleton) — (Case 16 — Mrs. C.) — Pedunculated orbito-ethmoidal exostosis projecting into the orbit.

Pedunculated Ethmo-orbital Exostosis

CASE 16 — WOMAN C. — Two years ago, following "a cold in her head," the right eye began to bulge without pain or diminution in vision. At the time of examination, the patient was not conscious of double vision, "excepting oc-

casionally." Red glass test, however, revealed a homonymous diplopia in all fields.

The eye was proptosed, being 3 mm. lower than the other. A firm mass could be felt at the inner and upper angles of the orbit.

The eyes of the patient's sister have a "tendency to bulge." Roentgen plates showed a "lobular bony growth which, apparently, fills most of the right ethmoidal region projecting into the orbit, but not involving the frontal sinus."

Operative removal of a pedunculated lobulated exostosis from the inner angle of the orbit, by drilling away the collar of normal bone through which the mass protruded into the orbit. As the sinus was approached, the tumor was seen to be covered by mucous membrane which could easily be stripped off the growth. Bacteria grew on culture.

Histological Examination (Dr. Lewis Brown): "The tumor is composed of numerous areas of new bone growth of varying size. The periphery of these areas shows considerable osteoblastic activity in the form of large osteoblasts. The surrounding bone is quite dense and the cells are few. Those which are present are rather small."

FIG. 12 — (Eagleton) — (Case 16 — Mrs. C.) — Lateral view of same showing growth filling the entire ethmoidal region and projecting slightly into the frontal sinus.

Piarachnoid Abscess as a Complication of Accessory Sinus Ivory Exostosis and its Frequent Recurrence

Frontal lobe subdural space abscess or piarachnoid abscess is very apt to be a complication of an ivory exostosis; and although the removal of a nasal sinus osteoma or exostosis is never followed by a recurrence of the growth, it never "metastasizes," the intradural suppuration which accompanies it may recur, and this several times. I have had one case in which the brain abscess reappeared eight years after the removal of the bony tumor, and again seventeen years after the original operation.

Meningoblastomata Above the Orbital Roof as the Causation of Exophthalmus.

The two varieties of growth of the roof of the orbit that are of surgical importance are (1) meningoblastomata (Mallory), Cushing's meningiomata of the sphenoidal ridge[40] which cause decalcification[41] of the bone, but which rarely invade the orbit at an early stage, and (2) the meningoblastoma of the olfactory groove. In former days, olfactory groove tumors were allowed to attain enormous size before being operated upon, when they frequently caused bilateral exophthalmus.

The Osteogenetic Character of the Special Sense Bones[42] of the Sphenoidal Ridge and Olfactory Groove Does Not Favor the Growth of an Overlying Cranial Exostosis of the Orbital Roof. Consequently, When Exophthalmus Is Present, It Is the Result of Invasion of the Orbit by the Tumor

It is to be noted that in meningoblastomata of the olfactory groove, exophthalmus does not occur until very late and after the growth has invaded the orbit; this because an overlying cranial exostosis, which is so frequently present with meningiomata of other parts of the cranium, does not occur in the orbital roof.

40. Bailey, Percival, *Intracranial Tumors*, Chas. T. Thomas, Baltimore, 1933, p. 176.
41. Camp, J. D., *Intracranial Calcification and its Roentgenologic Significance*, Am. J. Roent. & Radium Therapy, 23:615-624, 1930.
42. Eagleton, Wells P., *Infections of a Petrous Apex and of the Sphenooccipital Basis as a Cause of Suppurative Meningitis—A Study of Bone Growth in its Relation to Pyogenic Lesions, with Especial Reference to the Influence upon Infections of the Reticular, Histiocytic and Phagocytic Macrophagic Elements that are Contained within the Marrow of Bone* (in press).

I am of the opinion that this absence of exostosis over a meningoblastoma of the olfactory groove, or of the inner part of the sphenoidal ridge, is due chiefly to the special sense nature of the bones of this region. Kolodny[43] believes the bony proliferation of an overlying exostosis is an evidence of a defense reaction of the bone, because of the increase of its blood vessels.

Both the special sense (ophthalmic) portion of the sphenoidal ridge and the special sense respirato-olfactory bone of the olfactory groove are almost avascular from birth and, consequently, do not readily respond by proliferation, as does bone marrow-containing bone, although continued pressure in either location at first causes decalcification, and then, absorption. Exostosis superimposed on a meningioma is always accompanied by an infiltration of the bone itself (Penfield[44]).

On the other hand, a meningoblastoma which is situated well outward on the sphenoidal ridge may stimulate the medullary tissue around the pterion and produce diffuse thickening of the bone around the apex of the orbit; however, it but rarely occasions exophthalmus.[45]

Congenital Anomalies and Dehiscences of the Orbital Roof as a Cause of Exophthalmus

(a) Developmental anomalies in the orbital roof, such as in tower skull, may give rise to marked exophthalmus, or the roof may be the seat of (b) congenital openings through which cranial tissues (meningoceles or encephoceles) may project; or openings from (c) metabolic disturbances from lipoid dystrophy, such as in Schiller-Christian disease, may develop, any of which may occasion exophthalmus.

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43. Kolodny, A., *Cranial Changes Associated with Meningioma "Dura Endothelioma,"* Surg. Gyn. & Obst., 48:231, 1929.

44. Penfield, Wilder, *Cytology and Cellular Pathology of the Nervous System,* Paul B. Hoeber, New York, 1932.

45. David and Stuhl established a quasi-certain diagnosis of meningoblastoma of the lesser wing of the sphenoid in three of twenty-three cases, and in five others a probability, the most reliable signs being those indicating osteoma of the lesser wing, or of localized atrophies and calcification. (David, M., and Stuhl, L., *Roentgenologic Signs in Meningioma of Lesser Wing of Sphenoid,* J. de radiol. et d'électrol., 17:193-226, (April) 1933).

Thyrotoxicosis and Hypertension as a Predisposing Cause of Exophthalmus

Thyrotoxicosis or hypertension may predispose to exophthalmus. Thus, of the cases herein reported, one (Case 11 — Mrs. C.) had an enlarged thyroid several years before the cavernous sinus infection, and two others (Case 15 — Mrs. S. and Case 17 — Man B.) had hyperthyroidism some years before the tumor formation.

Influence of Trauma in the Production of Bone Tumors of the Orbital Walls

Traumatism certainly plays a large rôle in the development of bony growths of the outer walls of the orbit (Case 17 — Man B.), but not of the inner nasal surface, the bony tumors of which, as previously stated, result from infection.

Angio-endotheliomata Involving the Orbital Walls Associated with Disseminated Areas of Calcification Within the Skull

In one of my cases of angio-endothelioma (Case 17 — Man B.), the bone surrounding the orbit thickened. The calcification was so diffuse that ultimately isolated areas appeared in the falx cerebri.

Case 17 — C. B. — A young man, in 1927, fell and struck his nose. A year later he first noticed a slight swelling of the upper lid which gradually increased.

On March 23, 1929, he had a slight exophthalmus, the left eye being a little lower and more prominent than the other.

No annoying diplopia, but with a red glass there was double vision in the upper field. Veins of left fundus fuller than those of right side. On auscultation over the eye, no abnormality was heard, but pressure on the carotid produced alteration of the normal sounds.

The thyroid was slightly full and there was a fine tremor of his fingers.

The basal metabolism was normal. Roentgenograms at this time were negative.

Diagnosis: Tumor of optic nerve.

In April, 1931 (2 years later), the exophthalmus had increased markedly. There was a slight ptosis (see Fig. 13) and a mass about the size of a peanut could now be felt in the upper temporal angle of the orbit.

Auscultation over either eye revealed a bubbling noise; which was louder on the left side.

Pressure on the carotid had no effect upon the noise or the proptosis.

ROENTGEN EXAMINATION: There was now sufficient bone pathology to account for an exophthalmus within the left orbit. Dr. Charles F. Baker reported, "It is my impression that at the time of the injury there was a fracture

FIG. 13 — (Eagleton) — (Case 17 — C. B.) — Exophthalmus before operation.

which involved the posterior and outer portions of the left orbit and greater wing of the sphenoid, for in this area there is a dense white line three centimeters in length, extending well into the orbit and terminating in the middle third of the sphenoidal fissure. It is very opaque and, I believe, that it indicates callus formation. However, it may be due to thickening of the periosteum and not to deposits of new bone. Extending along the greater wing of the sphenoid, there are also shadows of increased density, rectangular in outline, which also suggest bone thickening." (See Fig. 14).

OPERATION: Kroenlein flap of left orbit exposed many nodular masses attached to the outer orbital wall. They were easily peeled out with but little bleeding.

HISTOLOGICAL EXAMINATION (Dr. Lewis Brown): "Shows a tumor composed of firm blood capillaries and fibrous tissue. The fibrous tissue is of the adult type. The blood vessels are numerous, with only slight activity of their lining cells. There is no evidence of malignancy."

DIAGNOSIS: Angio-fibroma.

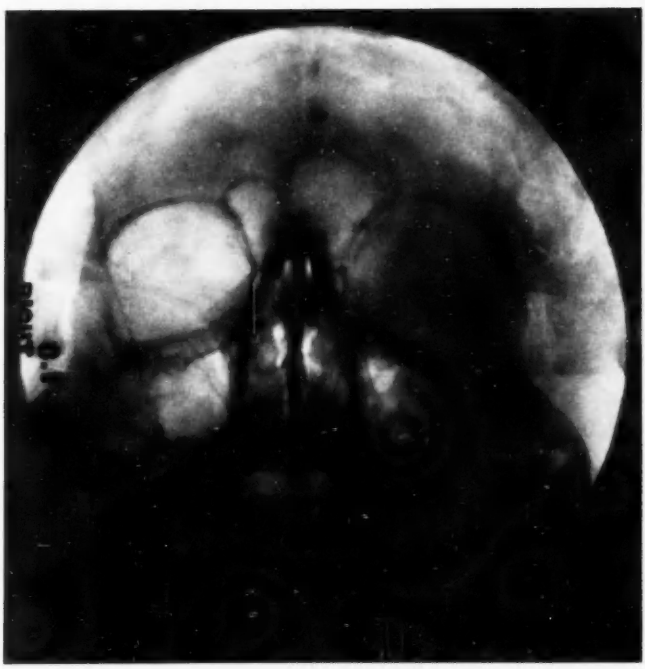

FIG. 14 — (Eagleton) — (Case 17 — C. B.) — General bony thickening of the orbit in a case of angio-osteomyomata.

POST-OPERATIVE RESULT: Immediately following the operation, the eye resumed an almost normal position (see Fig. 15); however, the veins of both fundi continued large, and the left disc was slightly pale.

After several months of freedom from all symptoms, there was a very gradual return of the exophthalmus; and two and a half years after the operation he was again unable to close the lid over the eye.

ROENTGEN EXAMINATION: In October, 1933, Dr. Baker reported: "Extending from about the middle of the vertical diameter of the left orbit, heavy deposits of bone density are seen extending laterally, outward and overlying the temporal fossa. These shadows are shaggy and of variable density, but have clearly defined margins. In the lateral view, this increased density extends backward along the skull to a point below the shadow of the sphenoid cells.

FIG. 15 — (Eagleton) — (Case 17 — C. B.) — Slight exopthalmus remaining after Kroenlein operation.

"The extensive new growth of bone appears to be of benign character because of its slow growth and the failure to show any erosion. It probably should be classified as an osteoma."

SECOND OPERATION: Extensive removal of the bone of the outer wall of the orbit, which was infiltrated by a gelatinous material. This allowed an exploration of the orbit, which was now found to be filled with diffusely and firmly encapsulated masses of tissue. The center of each mass was soft and easily enucleated (Fig. 16).

HISTOLOGICAL EXAMINATION (Dr. Lewis Brown): The section is composed of many small blood vessels whose walls show marked endothelial proliferation. These proliferating cells in places form masses in which no vessels can be distinguished. Histologically, the cells are only moderately active, but there is some evidence of a low-grade malignancy of the tumor.

MICROSCOPICAL DIAGNOSIS: Angio-endothelioma.

SUBSEQUENT HISTORY: The operation failed to relieve the exophthalmus; an ulceration of the cornea resulted from the swelling and operative trauma, following which the eye

FIG. 16 — (Eagleton) — (Case 17 — C. B.) — Roentgenogram after second operation, with extensive removal of bone. Note calcification in falx cerebri showing the diffuseness of the calcium reaction within the skull.

was enucleated. Radium treatment then caused a recession of the orbital tissue, so that the orbit is now carrying an artificial eye.

COMMENT: In the light of my present knowledge, I am of the opinion that the exophthalmus was not controlled and the eye subsequently lost because of failure to decom-

press the posterior part of the orbit sufficiently. This should have been done on both its lateral and cranial walls and, if necessary, on the inferior maxillary surfaces; for if I had relieved the exophthalmus by sufficient bony removal, the tumor of the soft parts might have been controlled by radiation and the eye saved.

Leontiasis Faciei

Although I have seen a number of these cases, none of them have been associated with exophthalmus (Dreisch[46]).

The Binocular Fusion Mechanism Has Remarkable Powers of Compensation. Consequently, Orbital Decompression for Exophthalmus Is Promptly Followed by the Restoration of Single Vision

Bony growths of the sides of the orbit, congenital bony deformities, as in tower skull, inflammatory swellings from the inner nasal sinus, or the lower maxillary, press the orbital contents forward, causing proptosis, which has the effect of decompressing the orbit. It is the exophthalmus which prevents injury to the nervous and circulatory mechanism within the retrobulbar spaces that are necessary for the proper functioning of the eye. Spontaneous decompression by the development of an exophthalmus is a protective measure, and so skillfully is the binocular compensation performed that when the exophthalmus appears gradually (such as in Cases 16 and 17), there is no accompanying double vision, except at intervals and in infrequent positions of the eyes.

Restoration of Binocular Vision by Paralysis of the Opposite Superior Oblique

On one occasion, a *frontal sinus operation* resulted in diplopia from the destruction of the bone to which is attached the "pulley" of the superior oblique. Following a similar operation on the opposite frontal sinus, two years later, the double vision immediately disappeared.

46. Dreisch, Cent. f. p. Augenh., 35:136; abstracted in Am. Ency. Ophth., vol. 7, p. 4857, Cleveland Press, Chicago, Ill., 1915.

SUMMARY

Summary of Surgical Infective, Neoplastic and Circulatory Diseases of the Cranial Cavity, the Nasal Sinuses and the Bony Orbit as the Cause of Exophthalmus, Especially as to Involvement of the Protective Retro-bulbar Space

(1) The orbit is a bony goblet-shaped cone, open in front.

(2) Posterior to the eyeball, the orbit contains a closed fascio-membranous muscular space — for which I would suggest the name, "protective retro-bulbar space."

(3) It is filled with fat in a semi-liquid state. Histo-functionally, it is a visual sense protective space, for it contains ectodermal elements and mechanisms for the protection of the eye from (a) circulatory disturbances from behind, as well as (b) acting as a cushion against external trauma. It (c) reacts uniquely to infection.

(4) The arterial supply within the retro-bulbar space is cerebral. The arteries spring from the junction of the vascular bed of the arachnoid base with its dural covering.

On the other hand, the venous return is diversified, the blood passing chiefly through the cavernous sinus; anteriorly escaping by way of the facial system, and, inferiorly, into the nasopharynx and the infratemporal fossa. Thus, a physiological increase in the amount of blood within the space is (a) promptly compensated for without displacement of the eyeball, (b) provided the veins and arteries possess their normal reactive contractile properties. However, any increase in the total amount of arterio-venous blood retained within the space results in exophthalmus.

Thus, the extreme exophthalmus of Graves' disease, which is out of proportion to the other symptoms of hyperthyroidism, and in which autopsy fails to disclose sufficient pathology to account for the proptosis in the orbital contents, is (a) uniformly relieved by an orbital decompression (Naffziger[47]); this (b) because the exophthalmus is due to an overfilling by blood of the retro-bulbar space from distension of the veins.

(5) Two-thirds of all cases of pulsating exophthalmus are due to an increased amount of circulating blood within the

47. Naffziger, H. C., *Progressive Exophthalmos following Thyroidectomy*, Ann. Surg., 94:582-586, 1931.

retro-orbital space, the result of an intracranial arterial venous communication.

(6) Sudden phlebitic invasion of the retro-bulbar space is characterized by diminished or (a) complete loss of vision, with (b) constriction of the arteries of the retina from "*stupeur artérielle*" (Leriche) (arterial coma). The fundus changes are apt to be associated with (c) haziness of the cornea out of proportion to the proptosis, and by (d) pupillary abnormalities (smaller pupil) (Case 2 — Boy W.).

(7) The whitish nerve head and the absence of retinal arteries present a different ophthalmoscopic picture than does embolism of the central artery of the retina, the usual cherry-red zone around the macula being absent.

(8) The blindness, with contracted arteries, is a protective reaction, due to the sudden effect of the infection upon the sympathetic nervous mechanism within the retro-bulbar space. A somewhat similar ophthalmoscopic picture, but without diminution of vision, is frequently seen immediately following a severe cranial trauma. This, however, is probably the result of a disturbance of the central sympathetic mechanism within the brain.

(9) The exophthalmus, chemosis, swelling of the eyelids and ocular fixation in cavernous sinus thrombophlebitis following an infection of (a) the face, (b) lip, or (c) nasal orifice, as a rule, is the result of a diffuse thrombophlebitic phlegmon of the orbital tissues. In this type of cavernous sinus infection, the fundus changes are limited (a) to distension of the retinal vessels, and there is (b) no reduction of vision until haziness of the cornea appears.

(10) "Blue eyelids," slight and transient proptosis, with or without chemosis, in the presence of a suppurating ear and a compensatory thrombophlebitis within an adjacent venous radicle is suggestive that the infection has involved the cavernous sinus. The orbital symptoms are due to a transient venous stasis from interference with the return venous flow from the anterior part of the cranial cavity. In these cases, chemosis and exophthalmus can be reproduced by the continued pressure in the neck from a sandbag during operation.

(11) Consequently, in exophthalmus associated with or resulting from a suppurative lesion of the face, nasal cavity, or ear, before exploration of the orbit, the surgeon should diagnose —

(a) Exophthalmus due to a localized orbital abscess following nasal sinus suppuration. Incision and drainage along the inner orbital wall promptly relieves the condition.

(b) Exophthalmus, the result of either a slowly or rapidly advancing orbital phlegmon, the causative lesion of which is diffuse endophlebitis of the orbital veins following a carbuncular infection of the lip, nasal orifice or eyelid. In diffuse phlebitic phlegmon, blind operative trauma into the orbit, in the hope of finding pus but accelerates the phlebitic infection. In the past, it has but too frequently converted a semi-quiescent orbital phlegmon into a fulminating diffuse lesion with an immediate cavernous sinus phlebitis.

(c) Slight or severe proptosis from infection of the protective retro-bulbar space is diagnosable by (a') sudden loss of vision, (b') whiteness of the nerve head, (c') contracted arteries, which may be associated with steaminess of the cornea out of proportion to and, thus, not accounted for by the amount of proptosis, and (d') delayed and impartial dilatation of the pupil of the homolateral side. These symptoms call for the immediate opening and drainage, not of the peri-orbital tissues, but of the retro-ocular space itself.

(12) Retro-bulbar space exploration and drainage are best accomplished through a latero-orbito-cranial approach.[48,49] If drainage is instituted early, it may prevent necrosis of the retro-orbital fat and an extension of the infective phlebitis into the cavernous sinus.

(13) An operation for any phlebitic process that involves the cavernous sinus should be preceded by a complete or fractional ligation of the common or internal carotid artery, because the ligation (a) places the inflamed cavernous radicle at rest, (b) relieves venous stasis, as well as (c) controls hemorrhage, which latter in cases of phlebitis is always so profuse as to prevent proper inspection of the parts and has resulted fatally in several instances.

48. Eagleton, Wells P., *A Case Showing the Possibility of Exploration of the Orbital Contents from Behind through the Cranial Fossa*, Arch. Ophth., 41:162, 1912.

49. Eagleton, Wells P., *Traumatic and Infective Lesions of the Head, the Chief Manifestations of Which are Visual Disturbances, their Diagnosis and Surgical Treatment*, Surg. Clinics North Amer., New Jersey Edition, (Dec.) 1926, p. 1441; S. Clin. N. Amer., (Dec.) 1926, 6:1435-1452.

(14) Several cases are herein reported in which carotid ligation alone has relieved the exophthalmus and, in one instance, restored vision to the eye.

(15) Slowly growing bony tumors of the orbit, which exert pressure outside of the fascio-muscular cone, causes but relatively slight displacement of the eyeball. Orbital decompression, if it is performed over the site of the maximum pressure, completely relieves exophthalmus. Consequently, in bony tumors of the orbital wall, complicated by exophthalmus, the orbital decompression should aim at complete freeing of the retro-bulbar space.

Section on Ophthalmology

The American Academy
of Ophthalmology and Otolaryngology

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Thirty-Ninth Annual Meeting
Chicago . . . 1934

DETACHMENT OF THE RETINA

REPORT ON OPERATIVE RESULTS IN 150 CASES*

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JOHN P. MACNIE, M.D.
(By Invitation)

NEW YORK

THIS PAPER is an analysis of the operative results on 150 cases of retinal detachment admitted to the Eye Department of the Presbyterian Hospital between October, 1930, and April 1, 1934. In this series, 197 operations were performed on 155 eyes; both eyes were operated upon in 5 cases. Since no attempt was made to select the cases, many so-called hopeless ones were included in this group. All cases in which the possibility of spontaneous cure had been eliminated were offered a chance by surgery. This attitude was assumed with the hope that we might learn by experience more about the indications for operating for this condition. With the same idea in mind, we have, in this paper, analyzed the failures as well as the successes. The operative techniques employed were the Gonin cautery puncture, the chemical cauterization as described by Guist and Lindner and the electro-coagulation method of Walker. A comparison of the results obtained by each method is given.

GENERAL STATISTICS

Sex — It is interesting to note that in this series of 150 cases 64% were males and 36% were females. These percentages are very close to those given by Shapland† in his series of 425 cases, his figures being 61.2% males and 38.8% females.

Age — The average age was 39.8 years, the youngest being 5 years and the oldest 75 years. The interesting feature in the age statistics is that in each decade between 20 and 60 there was no material difference in the number of cases encountered nor

* From the Institute of Ophthalmology, Presbyterian Hospital, New York.
† *British Journal of Ophthalmology*, Vol. XVIII, No. 1, Jan., 1934, pp. 1-23.

did we find that the age had any bearing upon the result of the operation.

Trauma — In these 150 cases, 45 of them gave a definite history of trauma and in 17 additional instances there was a history suggestive of injury, so we can say that trauma was a definite factor in producing the detachment in 30% of these cases and was at least a probable factor in 41.3%. Studying the frequency of trauma in relation to the refractive error showed that definite trauma was responsible for 41.6% of the detachments in hyperopes, while of the high myopes only 27.4% gave a positive history of injury. Of the 62 cases with a traumatic history 45.1% were either cured (30.6%) or improved.

Ocular Findings

No particular preference was found in the eye involved. In this series of 150 cases, the right eye alone was affected 64 times, the left eye alone 62 times, with involvement of both eyes in 24 instances. In 5 of the bilateral cases aphakia was present.

Refraction — A study of the refraction was undertaken to determine if possible the rôle myopia plays in the production of detachment. No definite information was obtained other than that myopia exists in 66.6% of the cases, while only 20.6% were either emmetropic or hyperopic. The detachment followed a cataract extraction in 12 instances (8%), but of these 5 were known to be high myopes, 2 moderately myopic (— 3.00 to — 6.00) before cataract extraction. In 7 instances (4.6%) sufficient data were not obtainable to ascertain the refractive state. The frequency of retinal detachment in myopia is well known, but it is interesting to note that Shapland found myopia present in 62.3% while, if we exclude the aphakics as he did, our percentage is 61.9%.

Refraction	No. of Cases	Percentage
+ to 0	38	25.31%
0 to — 3.00	20	13.33%
— 3.00 to — 6.00	18	12.00%
— 6.00 up	55	36.6 %
Aphakia with:		
(a) Hyperopia	5	3.33%
(b) Moderate Myopia	2	1.33%
(c) High Myopia	5	3.33%
Unknown	7	4.65%

Muscular Imbalance — It was our endeavor to determine what, if any, muscular imbalance was produced by the severance and reattachment of the recti muscles. From our data it would seem that postoperative diplopia is very rare as in only two cases was any note made of its presence; however, in 20 instances postoperative tests showed a slight paresis of the detached muscle. In 1 case an overaction of the reattached muscle was obtained requiring a prismatic correction for the resultant hyperphoria.

Intra-ocular Tension — In 55 of these cases where a careful preoperative and postoperative record of the intra-ocular tension (Schiotz tonometer) was available, the average tension before operation was 11.1 mm. Hg., while in these same cases the postoperative tension was 10.1 mm. Hg. The average postoperative tension in 50 cured or improved cases was 11.6 mm. Hg., while in 55 cases of failure the average postoperative tension was 9.5 mm. Hg. In 10 of these cases there was a postoperative hypotony of 6 mm. Hg. or lower whose average was 2.5 mm. Hg. If these are excluded from the failures the average tension of the others is 11 mm. Hg. This indicates that the operation itself has no appreciable permanent effect upon the intra-ocular tension. In 5 of these 55 cases a preoperative tension of less than 6 mm. Hg. was noted and in none of them was a cure effected, although in 2 instances the postoperative tension rose to 10 mm. Hg. or more. This is in accord with the general impression that profound hypotony is a grave prognostic sign.

Changes in the Crystalline Lens — Lenticular opacities developed postoperatively in 7 instances, yet in none of them was a satisfactory result obtained. Not a single successful case showed any clouding of the lens as a postoperative complication. The time elapsed since operation is, of course, too short to predict the future developments in these cases, but it is interesting to note that so small a number of even the failures have developed lenticular opacities. Those cases developing postoperative cataracts were all extensive detachments usually with profound hypotony.

Changes in the Field of Vision — In the vast majority of these cases the defect in the field of vision corresponded rather closely to the area of detachment. When a successful reattachment was effected the field for form usually returned to normal or near normal, although residual defects were frequently encountered. Refined perimetry is essential to elicit these defects,

but it is our opinion that if the tests are carefully done some impairment of function is demonstrable in almost all of them. The amount of impairment depends not only upon the preoperative viability of the detached retina but also upon the extent of the damage resulting from the operative procedure. A study of the fundus changes in the successful cases convinces us that the degree of reaction necessary for a cure must impair somewhat the function of the treated retina.

Relation of Time of Onset to Operative Result — A detailed analysis was made of this phase to determine, if possible, what effect a reasonable delay would have upon the result. To our surprise we found the results were just as good in the cases of over three months' duration as they were in those of less than one month's duration. The following figures show this rather strikingly:

Cases With Approximately One-Half of Retina Detached

Time of Operation	Within 1 month	1 to 3 months	3 months or more
Cure	23 (50.0%)	30 (48.3%)	16 (55.5%)
Improved	3 (6.5%)	3 (4.8%)	3 (10.3%)
Failure	20 (43.6%)	29 (46.8%)	10 (34.2%)

Cases With Approximately Three-Fourths of Retina Detached

Time of Operation	Within 1 month	1 to 3 months	3 months or more
Cure	5 (22.7%)	7 (23.3%)	4 (13.0%)
Improved	3 (13.6%)	3 (10.0%)	1 (4.3%)
Failure	14 (63.6%)	20 (66.6%)	19 (82.4%)

Cases of very long standing were too few to justify any conclusion, but suffice it to say that in 1 instance a reattachment with a return of the field was effected in a detachment of at least 5 years' duration. A study of these figures reveals that if a detachment is not progressing a reasonable delay does not materially lessen our chances. With this thought in mind, we next tried to determine whether the detachments invariably progressed. Many of the cases were not observed for any great length of time prior to operation, while in others the data were inconclusive. However, one gathers the impression from a study of these records that in frank cases progression is the exception rather than the rule.

Relation of Extent of Detachment to Result — In 97 of these 150 cases approximately ½ of the retina was detached and in these a cure was effected in 49.5%. In 36, with approximately ¾ of the retina detached, a cure was obtained in only 25%, and in 21 with an almost complete detachment a cure was gotten in only 9.5%. In 1 case, where only ¼ of the retina was detached, a successful result was obtained. From a comparison of these results it would seem that the extent of the detachment is a very important prognostic sign. One may argue, of course, that in the very extensive detachments, diseases of the retina, choroid and vitreous are more common. In spite of the truth of this statement the mere extent of the detachment seems to have an important bearing upon the outcome. Another important factor in reducing the chances of success in widespread detachment is the necessary extensiveness of the operative procedure. This leads one to wonder whether these cases would not do better with 2 smaller operations rather than with 1 very extensive one, which was tried in a few instances. This is merely a thought which we hope time will answer. A review of the cases requiring more than 1 operation shows over ½ of them were in detachments involving at least ¾ of the retina. Another very interesting thing is that while the outlook is poor in cases with an almost complete detachment 9.5% of them were cured. This should spur us on to further attempts at restoration of vision in these so-called hopeless cases.

Relation of Site of the Detachment to Result — Of the 97 cases with ½ of the retina detached we found the inferior half to be involved twice as often as any other part. The location of the detachment seems to have no appreciable bearing upon the outcome.

97 Cases With One-Half of the Retina Detached

Location	No.	Cure	Improved	Failure
Superior	22	10 (45.4%)	1 (4.6%)	11 (50.0%)
Inferior	48	23 (47.9%)	4 (8.4%)	21 (43.7%)
Temporal	21	12 (57.1%)	1 (4.8%)	8 (38.1%)
Nasal	6	3 (50.0%)	1 (16.6%)	2 (33.3%)

Relation of Type of Detachment to Result — A study of the type of detachment in these 97 cases with involvement of ½ of the retina was undertaken to see whether a bullous or a flat detachment responded better to surgical intervention. The figures are:

Type	No.	Cure	Improved	Failure
Bullous	81	40 (49.4%)	6 (7.4%)	35 (43.2%)
Flat	13	7 (53.8%)	1 (7.7%)	5 (38.5%)
Mixed	3	1 (33.3%)	0	2 (66.6%)

It can be seen by a study of these figures that most of the detachments were described as bullous, but that the percentage of cures and improvements is essentially the same for the 2 groups.

Relation of Holes to Result — In 86 (56%) of the 155 eyes one or more holes were found, while in 69 (44%) none were seen preoperatively. Furthermore, an attempt was made to determine whether the type of hole present could be of any prognostic value, so not only their presence but their nature was noted in relation to the result with the following findings:

86 Cases With Retinal Holes

	Irregular Tear	Round or Oval	Tongue	Dis-insertion	Multiple
Cure	20%	52.5%	46.5%	45.5%	42.1%
Improved	—	7.5%	—	4.5%	0.5%
Failure	80%	40.0%	54.5%	50.0%	47.4%

Of these 86 cases, we were able to benefit 46.4%, curing 41.8% and improving 4.6%, while in the 69 cases in which no holes were located the percentage of cures and improvements were 43.8% (cured 34.7% and improved 9.1%). In other words the "blind" operations were almost as successful as those in which holes were located and treatment concentrated around them. We do not wish to minimize the importance of sealing any existing holes, but rather to emphasize the curability of those in which no holes are found and to stress the necessity for widespread treatment in all cases. We were least successful in those cases presenting irregular tears, still one hesitates to draw any conclusion from so small a number. It is possible, however, that the difficulty in closing these irregular rents is responsible for the large percentage of failures in this group.

Description of Operations

Cautery Puncture (GONIN)

The retinal hole was carefully localized. At operation, the sclera in the region of the hole was exposed and a short linear incision made through it to the choroid. The cautery tip having

been heated to a dull red was inserted through the hole in the sclera and allowed to remain in the vitreous body 5 seconds. The conjunctival wound was closed with interrupted sutures and atropine 3% instilled.

Multiple Scleral Trephinings with Chemical Cauterization of the Choroid (GUIST-LINDNER)

The sclera in the region of the detachment was exposed, 1 or 2 of the recti muscles being cut if necessary. With a 1½ mm. trephine, openings were made in this area, the discs being dissected out to denude the choroid. The whole area of the detachment was treated in this manner, as many as 20 or more openings being made. The choroid was touched with a crystal of KOH for 5 seconds and immediately neutralized with ½% acetic acid, following which the whole field was irrigated with normal saline. Several openings were made in the choroid in dependent positions with a punctum dilator. Usually a copious flow of subretinal fluid was obtained. The muscles were reattached to their stumps, the conjunctiva was sutured and atropine instilled.

Diathermy (WALKER INSTRUMENT)

The sclera over the detachment was exposed as in the Guist operation. Platinum needles were inserted over the detached area, the current being so adjusted that perforation took place with very little pressure. A current up to 100 milliamperes and even higher was at times necessary. The needles were withdrawn following which subretinal fluid escaped. The wound was closed and atropine instilled.

Combined Operation

Some surgeons have combined the scleral trephine and diathermy operations. This was done for the purpose of obtaining better evacuation of the subretinal fluid.

With only a few exceptions the operation for detachment was done with general anæthesia, avertin with supplementary ether being used. In our experience there seemed to be as much postoperative nausea and vomiting following local anæsthesia as where general was used. It was suggested that traction on the globe during the operation was responsible for this in either

case, and as the operative procedures were more easily performed with the patient unconscious, a general anæsthetic was usually chosen. As soon as consciousness returned the patient was placed upon his back and kept in this position with as little as possible motion of the head for two and one-half weeks. If the detachment had occupied the inferior portion of the globe, the patient's head was slightly elevated, but was kept constantly in this position during this period. It was felt that this period of quiet was important.

Both eyes were bandaged at the conclusion of the operation and this dressing was changed for the first time on from the 3rd to the 5th day postoperatively and on every other day following until both dressings were omitted on about the 17th day. One per cent solution of atropine sulphate was instilled in the operated eye at each dressing and no attempt made to observe the fundus until usually the 9th day after the operation.

When both dressings were removed, on about the 17th day, the patient was allowed up by easy stages and both eyes were uncovered for a longer interval each day. The patient was usually ready for discharge about 21 days after the operation. The conjunctiva and muscle sutures were removed on the day before discharge from the hospital.

After leaving the hospital the patient was urged to lead a quiet life for a few weeks and to avoid any strains such as associated with lifting or jars.

Results

In the 155 eyes operated upon the condition was either cured or improved in 47.8% (cured 38.0%, improved 9.8%), while 52.2% were failures. Among these were 12 aphakics of which only 3 were cured. The cases were considered cured when the retina was completely reattached with enlargement of the field. The "improved" cases were those in which the field of vision became enlarged, yet some peripheral detachment persisted, or else the retina was in place but the field of vision was not materially enlarged. In all, 197 operations were performed on 155 eyes in 150 patients. Thirty-four patients had more than one operation, one of whom had 6, two had 4, seven had 3 and twenty-four had 2. In no instance was a patient benefited after 2 previous operations had failed. Of the 24 cases having had 2 operations, 5 were improved and 6 cured. In each successful

instance the first operation was followed by at least a temporary improvement.

In the cured and improved cases, practically all showed, preoperatively, opacities of the vitreous varying from a few fine to dense strands of opacities. Twenty-two cases showed pigmentary changes in the periphery with cystic degeneration of the retina. Three cases presented the picture of an old exudative choroiditis and in 5 hæmorrhages into the retina were found. In 1 case a rupture of the choroid was associated with the detachment.

Visual Acuity in Cures — In 56 cures the visual acuity before and after operation was studied. In 5, the acuity was worse after operation, however, in 4 of those the patient was able to read the next larger line on the test chart than he had before operation. In 1 case, vision fell from 20/30 to 20/100. In 29 cases, final vision was 20/50 or better, while in 20 cases it was 20/40 or better, being improved to this from as little as 3/200. In 47 cases, the vision was 20/200 or better after the operation.

Poor central vision usually could be attributed to preoperative retinal disease — myopic changes frequently being the cause. In one case a typical hole in the macula was found. It is our impression that once the macula has become detached restoration of central vision does not follow reattachment of the retina.

Comparison of Results with Different Operative Measures — The actual cautery operation as described by Gonin was done in 6 cases and they all resulted in failure. In each of these cases an attempt was made to localize and seal the hole. It is possible that improper technique was responsible for our failures, but great care was exercised in the selection and study of these cases as well as in the performance of the operation.

In attempting to compare the results obtained by the different techniques, it was thought to be fairer to include only those cases requiring one operation, as, often times, if one technique failed another was tried, making it difficult to determine to which method to attribute the result. Those cases on which only one operation was performed numbered 120. The Guist-Lindner method being used on 92, the Walker diathermy on 18 and the combined on 10. The chemical cauterization gave 46.7% cure, 6.5% improved, 46.7% failure, while the electro-coagulation method resulted in 38.8% cure, 16.6% improved, 44.5% failure.

The total percentage of those benefited by these two procedures was essentially the same, although the cures were greater in the Guist-Lindner group. The larger percentage of improved cases obtained by the diathermy method may possibly be due to too conservative treatment, which, if a little more radical, might have resulted in a complete reattachment.

Analysis of Failures

In the 81 cases in which the operation was not a success the attempt was made to determine the causes of failure. In 20 cases, no reason could be determined.

Of the conditions existing before operation which may have contributed to failure the commoner were the magnitude of the detachment in 42 cases and the presence of high myopia in 26. Atrophic changes in the retina and choroid were noted in 12 cases and hæmorrhages in the vitreous and retina were observed in 9. Inflammatory changes were seen in the retina and choroid in 2 cases, an old uveitis in 1 case and a retinitis proliferans in 1. Marked arteriosclerosis and vascular hypertension were present in 2 cases. There were 3 cases of intra-ocular foreign body in the series of 150 patients, in 2 of which it had been removed before the diagnosis of detached retina and in the remaining 1 an attempt at removal had been unsuccessful. In 3 cases, a preceding Gonin operation had led to scar formation in the retina and the development of traction bands in the vitreous. In this group there was also 1 case each of glaucoma with a corneo-scleral trephine operation 6 months previously, a large coloboma of the choroid, a question of metastatic carcinoma, epilepsy, microcornea with high myopia and a dislocated lens in a case of high myopia.

In 8 cases in which the Guist operation was being performed premature perforation of the globe occurred, resulting in loss of vitreous and an unsatisfactory result. Overtreatment leading to prolonged inflammation of the eye or definite injury was present in 4 cases.

Postoperative vitreous hæmorrhage was considered to result in failure in 9 cases and a uveitis in 7. In 7 cases the retina was in position on discharge from the hospital but redetached subsequently. The longest interval at which this occurred was 3 months. In 7 other cases the area of detachment increased after the operation and in 5 the presence of profound hypotony was

thought to be responsible for the poor result. In 1 case each retinal hæmorrhage, poor postoperative coöperation of the patient and the development of retinitis proliferans contributed to failure.

There were 9 aphakic eyes which were operated without improvement. Six had had routine cataract extractions from 2 to 15 months before the operation for detachment. In 1 of these the diagnosis of detachment was made 4½ years before. The lens was extracted and then the detachment operated without improvement; in 1 a loss of vitreous at the time of the extraction was noted. One case had had a linear extraction 8 years before, and 2 had had discissions 3 and 19 years preceding the detachment operation.

Of the failures, 3 cases finally came to enucleation and 1 was eviscerated, giving a percentage of 2.5 in the whole series in which the eye was lost.

Summary

In this series of 150 cases of retinal detachment 64% were males and 36% were females. The average age was 39.8 years, the oldest being 75 and the youngest 5 years of age.

A positive history of trauma was obtained in 30% of these cases with an additional 11.3% giving a history suggestive of injury, making a total of 41.3% in which trauma was at least a probable etiological factor. Some degree of myopia was present in 66.6%, 36.6% having 6 or more diopters of myopia, only 8% of the entire group were aphakic.

The severance and reattachment of the recti muscles rarely produced any noteworthy muscular imbalance.

The intra-ocular tension was not permanently affected by the operation. Marked hypotony was found to be a very grave prognostic sign.

Postoperative changes in the crystalline lens were rarely noted.

Some impairment of the field of vision was commonly seen after a successful operation.

Detachment of approximately ½ of the retina was cured in 49.5%, while in those with approximately ¾ of the retina detached only 25% were benefited.

The inferior half of the retina was detached about twice as frequently as any other part.

Those cases in which no holes were found responded to operation about as well as those in which one or more holes were located.

In the 155 eyes operated upon, the results were: cures 38%, improved 9.8%, and failures 52.2%.

The results obtained with the chemical cauterization method of Guist and Lindner were just as good as those obtained by the electro-coagulation method of Walker. Widespread treatment of the affected area seems to be necessary to effect a cure.

We wish to express our sincere thanks to the following members of the eye staff of the Presbyterian Hospital for their kind coöperation in allowing us to include their cases in this report: Doctors John M. Wheeler, T. H. Johnson, D. B. Kirby, A. B. Reese, G. M. Bruce, and H. S. McKeown.

Some of the cases included in this series have already been reported by Dr. H. S. McKeown* who graciously allowed us to reanalyze them for this report.

DISCUSSION

Dr. Clifford B. Walker, Los Angeles: The authors of this paper are to be congratulated on having presented us with such an excellent and fruitful study of perhaps the largest series of separated retina cases so far gathered together in this country. In my smaller series of 35 or 40 cases, I find much to confirm and sometimes even amplify the findings in this paper.

First, as regards the almost 50% greater frequency among males in my series seems to indicate a more strenuous life, with greater hazards and more frequent injury to the male than to the female. The actual causative trauma is often difficult to trace because defective central vision may not result till several weeks, perhaps months, later, at which time the traumatic incident may have been forgotten.

A study of the Lindner celloidin models has led me to believe that the types of injury most easily forgotten are not direct ocular injuries but remote jolts or jars that may produce a rotary jerk of the eyeball, which

* McKeown, H. S., *Detachment of the Retina: The Guist Operation and Report of Cases*, Arch. Ophth., new/ser., 9:64, (Jan.) 1933.
McKeown, H. S., *Detachment of the Retina: The Guist Operation*, Trans. Am. Acad. Ophth., 1932:240.

I have theorized must come more readily by transmission of the shock unequally to the third, fourth or sixth nerves, or their centers. Tears are frequently found in the neighborhood of the muscular attachments, especially the superior oblique.

In the dark, I have often looked suddenly in different directions to note the flashes or phosphenes which I fancy come from the region of muscular attachment. Judging from gelatin models, I believe the whip-snap from this cause to be greater if the tension or turgescence of the vitreous is low. Under such conditions, it seems to me a separation, even without a detectable tear, seems possible.

The authors have pointed out the well-known frequency of low tension before and especially after the operation and its grave significance. This is a very valuable part of their study. It seems to indicate a loss of vitreous turgescence and a dehydration through capillary dialysis and other portals of exit as a fundamental part of the necessary setup in many cases of non-contusive and even non-traumatic separation. This condition in many ways is quite the opposite to that in glaucoma.

The fact that there is not much depreciation in the statistical possibilities of operative cure during the first three months should not be used to encourage delay, because, in the first place, the detachment is often found to be of longer duration than the first history indicated. Then, again, if the patient gets some improvement by immobilization, it is sometimes difficult to prevent the patient from delaying until still worse statistical possibilities present themselves. Further, while the number of reattachments may be similar, refined examination may show the quality of the results to be of lower grade.

With regard to the question of two-stage operations, I believe that if it is necessary to barrage more than one-half the eyeball, it is better to attempt a two-stage operation. In one case, I was successful in secluding nine tears distributed over 185 degrees and requiring the use of 75 micropunctures, and two muscles were temporarily tenotonized.

It seems to me that gradually we will classify at least the traumatic cases still further and be able to offer more definite percentages of chance for reattachment and cure to each group. Already aphakia is separately classified with a low rating. Our authors have cured one out of four, which is twice as good as Safar's first series of cases indicated. I have cured two out of three and know of at least two other operations with about the same average, by micropuncture. I have operated successfully on one case of congenitally dislocated lens with recently separate retina surrounding the lens in the lower half of the globe. Boxers rate still lower. I have cured only one out of three, while tennis ball injuries in young people, I believe, can be rated above 60 per cent. But I have had no success so far with two baseball injuries, yet in two cases of detached retina from a single blow with a bare fist, both were cured, although one had three operations and the other had not only a peripheral tear but also a hole in the macula.

Hole in the macula is probably best treated at present by the Lindner undermining with KOH injection method, starting from just above the

inferior oblique attachment as the site of election. On four eyes, six operations were done with 50% success. I can agree with Lindner that, if this operation is not successful the first time, it is not likely to be cured by a repetition of the operation.

Massage of eyeball, as performed by chiropractors or masseurs or by a night-worker in the effort to keep awake, may, in older people, produce a separation. In two cases I obtained a primary reattachment but one separated later and refused further operation.

I wish to thank the authors for giving my operation such a careful trial and feel quite gratified to note that it compares favorably with the results obtained by their unsurpassed skill in performing the Lindner-Guist operation. In less experienced hands, I believe, however, the micropuncture diathermic operation can be performed with no risk of losing good vitreous and with correspondingly higher percentages of cure. Loss of vitreous through a trephine opening results in failure, as a rule. If this happens eight times in a series of 100 cases, statistics would indicate that about four or more of these should have been cured by the micropuncture operation. This considerable handicap, even in the most skillful hands, will probably suffice to bring the micropuncture procedure into still greater favor.

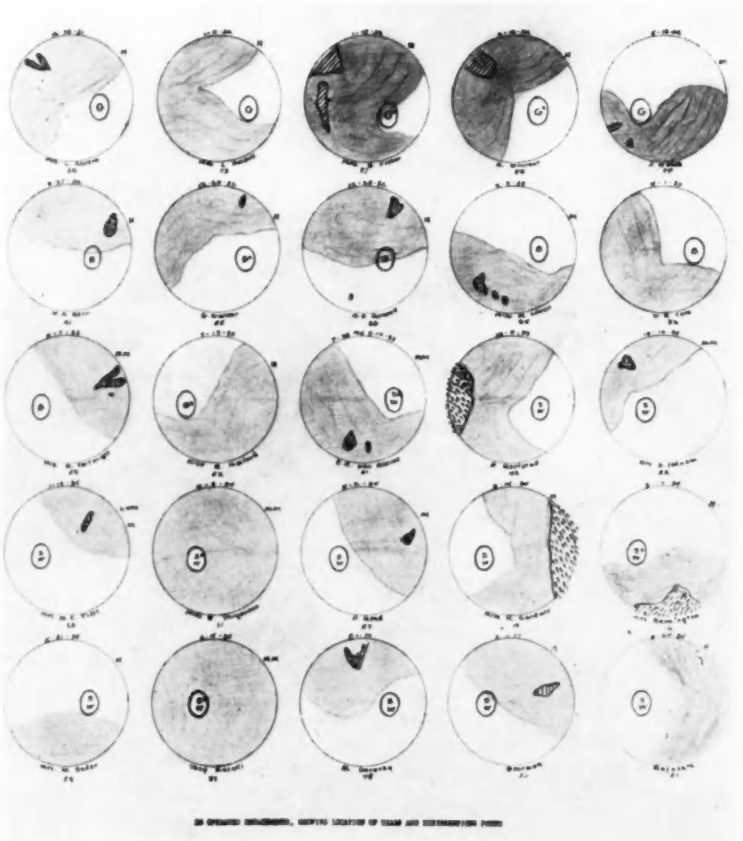
However, the Lindner method of trephining and undermining subsclerally to the macula in case of holes or tears near the posterior pole still has no real competitor. In this procedure $\frac{1}{50}$ cc. of 4% KOH may be injected through a 2 mm. linear or a 1¾ mm. trephine opening [with button only half elevated or hinged and not removed] 20 to 26 mm. back of the limbus, preferably just above the attachment of the inferior oblique. Here ophthalmoscopic control is easiest, but any other part of the temporal side may be used with considerable success.

Again, I wish to congratulate the authors for their extremely valuable contribution to our knowledge of this subject.

Dr. F. E. Burch, St. Paul: The Academy is indebted to the authors for so fairly analyzing their results, and giving the causes of their *failures*. This is the first *large* collected series of operations for retinal detachment to be reported on this side of the Atlantic.

I would like to mention two instances of disinsertion downward following trauma, with retinal detachment in the lower quadrant, which healed without operative interference. Rest in bed, with semi-recumbent position, atropinization and *occlusion of both eyes* to prevent ocular excursions, obviated the necessity for operation and resulted in perfect reattachment. Perhaps this occurs more frequently than we know, owing to the fact, that disinsertions invariably occur downward or temporalward. When it occurs as a result of trauma, the vitreous is often healthly, and conditions are favorable for spontaneous healing. Where moderate hemorrhage occurs accompanying these disinsertions, undoubtedly the agglutinating properties of the blood may sometimes be helpful in securing reattachment.

The authors report a remarkably high percentage of successes following the "blind" operation. Failure to close or wall off the retinal hole is the largest factor in unsuccessful results, in my experience. With 25 operations, no hole was found in 7 instances, and operation failed in each of these cases. Of the remaining 18 in which definite holes or disinsertions were found, good results were obtained in 13 cases. This leads to the very definite conviction that finding and properly localizing a retinal hole, tear or disinsertion is almost absolutely necessary for good surgical results.



As a means of discovering retinal holes, I would like to emphasize the importance of absolute rest in bed with complete occlusion of both eyes. One may ofttimes thus discover a hole, after the retina has become flattened down, when it could not be found at previous examinations. As is shown in the diagram of my operated cases, the predominance of holes

in the superior quadrant is quite evident. Excluding the disinsertions, holes were found in 15 of the other 22 patients. In 11 of these 15, or 73.3%, tears or holes were exclusively in the superior part of the retina, slightly more often in the temporal than in the nasal quadrant. Contrary to expectations, the results proved slightly better in upper quadrant detachments with holes than when these occurred in the lower fundus, but possibly because in the majority of them the detachments were of more recent origin. Detachments in the upper pole are always discovered more promptly than when they occur below.

All of my operations were done under local anesthesia, retrobulbar anesthesia in 21 cases. Using morphine and scopolamine, I have found that retrobulbar novocaine injections provide entirely satisfactory anesthesia, and there is rarely any postoperative nausea. Pantocaine should be substituted for cocaine, because of desquamation of the corneal epithelium and cloudiness where cocaine was used, which makes it very difficult to view the fundus and relocalize retinal tears at the time of operation.

The contributors to the authors' series met with no success in any of their Gonin operations. In my series, 5 (all recent detachments) were done by the Gonin method with 1 favorable result. One was successful for 10 weeks, but became redetached after a fall.

I can make no comparison between the results of the Guist and Safar operations. After I had seen the Guist trephine barrage operation with chemical cauterization, the method did not appeal to me, and operation by this method was not attempted. In its stead, a barrage of electrocauterizations around the site of the tear or hole, where these were found, otherwise over the most prominent area of detachment, with one or more perforating cautery punctures was tried in 7 cases. Of the 7 cases operated by this manner 4 (57%) were perfect results, one was improved. In 2, where no tear or hole could be discovered, the operation failed utterly.

Thirteen patients were operated by the Safar method, using the Walker high frequency apparatus, and the Walker iridoplatinum needles. Three of these were disinsertions. In 5 out of 6 patients where a hole was found, the results following diathermy are perfect *to date*. The results were excellent in the 3 patients with disinsertions. In 2 patients with total detachments of very short duration, no holes could be found. Each was operated in 2 stages, and no results whatsoever were obtained. In one patient in whom no tear in the retina could be found (after preliminary aspiration of subretinal fluid) we succeeded in securing a walling off of the detachment, but the detachment itself still remains, although flattened. In the 2 remaining cases, the results are promising, but are too recent to be counted as cured. As a result of experience with the Safar method, using the instrument and the needles designed by Dr. Clifford Walker, I have found technical difficulties are so simplified and the possibility of traumatizing the two most important structures involved, namely the retina and the vitreous, is so minimized, one may only conclude that the comparison with previous methods is entirely favorable to the Safar method.

In my series, 12 were females, 13 were males. Average age, 54. Trauma was a positive or suggested factor in 28%. Cures and improvements were about the same percentage in the traumatic and in the non-traumatic cases. Of the 25 cases, 7 were myopes, 5 were high myopes (over 4 diopters), 10 were hypermetropes, 3 unrecorded. Two developed complications; one developed iridocyclitis after a second operation, necessitating enucleation; one developed an infection in a reattached muscle tendon without impairing the result. The total number of cures is 13, or 52%; 2 were improved, bringing the total of *cured and improved* to 15, or 60%.

Of the 13 patients operated on by the Safar-Walker method, 8 were successful, with 2 more apparently cured but operated too recently to be included. However, the results in my small series are not actually as good as in the large series reported by the authors, for the reason that my patients were carefully selected; in the large series reported by the authors, as I understand it, they operated on practically *all* cases. I have refused operation to patients with very extensive detachments, and sometimes when unable to find a retinal tear. Also few operations were undertaken for very old detachments; none in detachments which followed removal of cataract.

President John M. Wheeler, New York: If my memory serves me correctly, about twenty years ago our fellow member, Dr. Jones, operated successfully on a case of detached retina by the cautery puncture method. I wonder if Dr. Jones will say a word and corroborate my statement.

Dr. Emmet L. Jones, Cumberland, Md.: I am very grateful to Dr. Wheeler for giving me this opportunity to bring to light a matter that has been very carefully buried ever since it happened.

In 1913, I operated by galvanopuncture on a man who was practically blind from detachment of the retina. I waited two years to see whether the result lasted, and the result was still holding good.

I published it in the Annals of Ophthalmology in 1915 under the title of "Detachment of the Retina cured by Galvanopuncture Combined with Massive Doses of Mercury Cyanide Subconjunctivally."

I had a lot of reprints, and I felt that there was no use in sending them out, that nobody was going to pay any attention to them, and they would just say, "There is one more fool advertising his folly." I let those reprints lay and turn yellow with age. In the course of time, events showed that it wasn't such a folly or such a fool after all.

Last year I sent them out with a little sticker on them to attract attention, that it would probably be of historical interest, and that the patient when last seen still had good vision.

That happened in 1913, and last year certain friends who were in frequent contact with this patient got from him emphatically that his sight was still holding perfectly good. That is twenty-one years ago, and the man is now seventy-six or seventy-seven years old.

So, in point of longevity or how long it might last, that, up to the present, is probably about the best we have got on this side of the water — twenty-one years.

Prof. Dr. Hans Lauber, Warsaw, Poland: I am perfectly of the opinion of Dr. Burch, that it is one of the most important things to find the hole. The looking after the hole is something which must be tried for a long time, sometimes. Of course, it is very important to keep the patient quiet. We generally give him the Lindner spectacles with the little hole in front, so that the eyes are kept quiet, without any pressure being exerted upon the globes.

We find very frequently that in a few days the retina detachment will flatten out and we can sometimes find holes which were invisible before. But I believe that one of the very important features in seeking for the hole is to have very intense illumination. We are using, at my suggestion (I suggested it a few years ago), a 3000-watt lamp which gives us 6000 candlepower. The light of this lamp is focused by a lens upon ground glass. That is what we use for the source of light.

We have found that by using this light, either in the indirect or in the direct method, with the usual mirror ophthalmoscope, we can locate holes in the retina which are practically invisible by other light.

I would strongly recommend to men who want to work in the line of retinal detachment surgically, to avail themselves of as powerful light as possible. I have sometimes followed the suggestion of Kruppa in using an arc lamp without any filter for quite a short time to look at those parts of the retina which seem suspicious. If we locate them in this way we will be more prone to find holes and then, also, to heal them.

In my place we have been going according to the same line, operating on all cases without any selection, to see what help we can give the patients and gradually to find out which cases were working better and which were working less well.

Our average good results have movements of about 50 to 55%. I believe that the best technique is to combine the Safar method of diathermic puncture and sometimes adding to it a slight galvanopuncture in place of the perforation of the retina.

Dr. Raymond Emory Meek, New York: In the past two years it has been my privilege to operate 21 cases of detached retina on the service of Dr. Webb W. Weeks at the New York Eye and Ear Infirmary. Since 7 of these cases I have been unable to follow, my thought at first was to omit them. The average age of these 21 patients was 44.5 years, and the duration from 6 days to 2½ years. Six of these were almost complete detachments — the remaining partial of varying degrees. In most cases there was some complication, 64.3% being myopic. Trauma is not considered as I could get a history of trauma in every case.

Nineteen of these cases were performed with "Gonin's" ignipuncture Two of the cases were electro-coagulation or "Larrson" operations, using

from 10 to 15 needles with the lowest current possible to enable me to penetrate the sclera, about 30 milliamperes. One of these cases gave me an excellent result.

Of the other 19, 7 were improved and 3 I consider cured. That means their fields were restored and that they regained useful vision and saw as well after operation as before the detachment occurred.

Of all the 21 operated, 4 were cured, giving a result of 19.4% cured.

One of these cases had a recurrence after 4 months and was operated a second time to give me a perfect result. The most recent of these cases was successfully operated 8 months ago.

DR. JOHN P. MACNIE, closing: We wish to thank those who have discussed this paper.

In regard to the time between the occurrence of the detachment and the operation, we feel that that is not as important as has been thought. A reasonable delay to enable the patient to get to general condition is perfectly justifiable.

We agree with Dr. Burch that the treatment should be concentrated on the region of the tear, but we feel that in those cases where no tear is found, the patient should be given a chance of operation.

We were very glad that Dr. Burch called attention to the fact that our cases were not selected. We operated on all who would allow us to do so.

THE USE OF SCLEROSING SOLUTIONS IN OPHTHALMIC THERAPEUTICS*

MORIE FREDERICK WEYMANN, M.D.

LOS ANGELES

INSTANCES of the use of sclerosing solutions for the treatment of ophthalmological conditions are rather rare in ophthalmological literature.

In 1925, Raverdino[1] reported the treatment of a patient who suffered with intermittent exophthalmos due to a varix of the orbit by injection of a solution consisting of four parts of quinine hydrochloride, two parts urethane, and thirty parts of distilled water. He had the patient hold his breath to produce a stasis of the circulation of the varix and then passed a needle to a depth of three centimeters along the inferior wall of the orbit. In order not to inject the solution directly into the lumen of a vessel the point of the needle was moved about until no blood was obtained on aspiration. Then 0.2 cc. of the above-mentioned quinine urethane solution was injected into the orbit following which there was severe pain for about five minutes. After this the patient was able to go home, but there were intermittent pains during the night. The right side of the face became swollen with dilatation of the facial and orbital veins. The next morning outward rotation caused some pain, but after the application of cold packs only slight discomfort remained. After two days this patient appeared normal except that the facial veins were visible under the skin. These increased in size on holding the breath. One week later this injection was repeated but this time the pain was very much less. Following the second injection there was gradual improvement until after six months the patient was entirely cured. This writer stresses the importance of injecting the solution into the orbital tissue outside the lumen of the vessel and states that the optic nerve was not damaged by the injection.

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Weekers,[2] in 1933, cited the case of Raverdino as a precedent and reported one of his own. His patient was thirty-seven years old and had an orbital varix in the left eye which followed a blow at the age of eight years. Varicose veins could be seen in the lower cul-de-sac. Under excitement or holding the breath a marked exophthalmos of the left eye occurred. Radium therapy was used without improvement. After the injection of 2 cc. of novocain into the lower outer portion of the orbit to a depth of three centimeters, 1.5 cc. of the above quinine urethane solution was injected at the same point. The next day the reaction was such as to produce an ecchymosis of the face and lids on the left side. This disappeared in fifteen days. The varicose veins in the lower cul-de-sac vanished, and a slight enophthalmos was produced so that the eye never protruded farther than the other eye even on jugular compression. The cure persisted when the patient was observed four and one-half years after treatment.

Weekers called attention to the observation of Bensaude and Oury that an endophlebitis and periphlebitis is caused by the injection of this quinine solution in the connective tissues surrounding the vessels without actually having it enter the vessels themselves. By this means he explains the beneficial effect obtained by injecting the solution into the orbit without attempting to gain access to the vessel lumen. He concludes that sclerosing injections constitute the treatment of choice in orbital varicocele.

A different use for sclerosing solutions was reported by Terson,[3] in 1931, where the subcutaneous injection of a five per cent solution of quinine di-hydrochloride and urea successfully overcame a spastic entropion. In a case of senile ectropion, the same writer injected 0.5 cc. of the solution submucously in the inner third of the lower lid and 0.25 cc. subcutaneously at the outer angle. The scar tissue thus produced gave just as good an effect as if operation had been performed.

The above cited instances of the use of sclerosing solutions show that those solutions which have their sclerosing effect due to quinine have been preferred. Perhaps this is because these solutions are in particular vogue in France.

From an analysis of the general literature on sclerosing solutions, which has become quite voluminous in recent years, an attempt may be made to determine which ones could be applied to ophthalmological therapy.

The principal use today of sclerosing solutions is in the treatment of varicose veins. In 1917, Sicard[4] noticed the obliteration of veins after injection of arsenicals for the treatment of lues. This focused his attention on a chemical method of obliterating veins and after considerable experimentation he found sodium salicylate to be most satisfactory for this purpose. However, when this salt is injected outside the vein severe necrosis occurs, and it is no longer used today by most operators.

In 1922, Genevrier[5] published an observation that in the treatment of malarial patients by the intravenous injection of quinine that if too great a concentration of solution were used thrombosis of the vein occurred. This would result in its obliteration. He was using a solution of quinine hydrochloride and urethane. Therefore, in France, this solution of quinine and urethane and also five per cent solution of quinine and urea became popular for use in the obliteration of varicose veins. These solutions also may cause necrosis if injected outside the vein.

More recently many other solutions have been tried for producing sclerosis of varicose veins, amongst them being high concentrations of the various sugar solutions and some of the salts of fatty acids, such as sodium morrhuate.

Ochsner and Garside[6] have tried experimentally the effect of injecting these various solutions into the veins of dogs, and examined these veins histologically after treatment. A forty per cent solution of sodium salicylate produced the greatest number of thrombi. The endothelial destruction caused by quinine and urea was far greater than that of concentrated dextrose solution.

Kilbourne, Dodson, and Zeiler[7] found that 0.5 cc. of the quinine urethane solution as used by Weekers was not fatal to rabbits weighing two kilograms, but that 0.7 cc. caused death. In testing the slough producing properties on the ears of rabbits it was found that sodium salicylate and quinine urethane solutions both produced larger and more persistent sloughs than sodium morrhuate. However, because of the greater potency of quinine urethane, in therapeutic doses it would have less tendency to produce sloughs than sodium salicylate. A solution of 0.5 cc. of quinine urethane injected subcutaneously in the human leg produced no slough. This was confirmed by injections of similar amounts in the writer himself. The quinine urethane solution used was found to be bactericidal with staphylococcus aureus and was used without sterilization.

From the fact that the quinine urethane solution had been used in ophthalmic therapeutics by previous investigators, that it possesses the property of obliterating vessels without being injected into the lumen, that it acts as an anesthetic after injection and is, therefore, less painful than sodium morrhuate, and that its sclerosing power is high in small amounts, further literature upon the application of this solution was investigated.

From a series of yet unreported experiments, Kilbourne[8] stated in a personal communication that a one per cent solution of quinine with urethane produced only seventeen per cent of sloughs as compared with fifty-six per cent with a one per cent solution of quinine with urea by the rabbit ear method. Where the quinine urethane solution was allowed to age the percentage of sloughs was reduced to zero. This would seem to indicate that some combination between the urethane and quinine occurred in the older solution.

In the application of quinine and urethane solution to the eradication of vascular tumors of the skin of both the cavernous and spider type, Andrews and Kelly[9] have reported the treatment of twenty patients. The solution consisting of four parts quinine, two parts urethane, and thirty parts water was diluted by them from one to six times, according to the tolerance of the patient. They also used a fifty per cent dextrose solution which gave about the same results as the quinine urethane. No attempt was made to inject the solution into a vessel, although this sometimes occurred. Hemostasis with a metal ring was advised during injection. Not more than 0.12 cc. of the quinine urethane solution was injected at any one point. There was some pain at the first injection, but none thereafter, and the injections were repeated at weekly intervals until the entire tumor was sclerosed. It is important to note that they obtained good results whether or not the solution was directly injected into a vessel.

So far, the discussion has concerned itself only with the obliteration of varicose veins or vascular tumors with the sclerosing solution. The application of this solution for the obliteration of cystic cavities was made by Kilbourne and Murray[10] where they succeeded in curing hydrocele by injecting two to four cc. doses after aspiration of the hydrocele fluid. They found that this injection also acted as an anesthetic so that when the fluid reformed, a second injection of 3 to 10 cc. of 13.66 per cent quinine di-hydrochloride, which has a greater sclerosing effect, could be used. Further injections were given at three-week intervals

when necessary. Some patients so cured were observed for more than one year.

My attention was called to the possible use of sclerosing agents in ophthalmological therapeutics when a girl six years of age, who had a cyst in each lower fornix, came under my observation. There was a history of a bilateral streptococcus conjunctivitis in early infancy. Recovery was uneventful. At the

FIG. 1 — (Weymann) — Right eye before injection (August 3).

FIG. 2 — (Weymann) — Left eye before injection (August 3).

age of three years, the parents noticed a growth in the lower inner corner of the left eye. Shortly thereafter, a similar but smaller tumor was noticed in the right eye. Examination of the child showed the visual acuity to be normal in both eyes

with no pathological changes in either globe. There was some scarring of the conjunctiva in each lower fornix with the formation of pseudo-symblepharon. In the lower cul-de-sac of the right eye in its mid-portion there was a conjunctival cyst about one centimeter long and four millimeters in diameter (Fig. 1). In the left eye a similar cyst extended from up behind the plica to beyond the mid-portion of the lower lid (Fig. 2). This cyst showed two constrictions at its nasal end which might have been interpreted as the position of the valves in a dilated lymphatic.

Because no tissue was removed for pathological examination, one could only speculate as to whether the origin of the cysts was from pockets of conjunctiva sealed off by the conjunctival inflammation, as in the formation of a pseudo-gland, or whether they were true lymphoceles. In any event, they would be lined by a thin layer of cells, whether of an endothelial or epithelial nature would be dependent on the origin. With the work of Kilbourne and Murray in the obliteration of hydrocele sacs in mind, and because the parents wished to avoid the use of a general anesthetic, it was decided to inject these cysts with quinine urethane solution. Accordingly, on August 3rd, under cocaine instillation anesthesia, two minims of a solution consisting of four parts quinine hydrochloride, two parts urethane, and thirty parts water was injected into the cyst in the right eye. The full strength solution was used because it was thought that the contents of the cyst would dilute the solution about one-half. There was immediate whitening of the collapsed cyst and a small amount of transient pain to the patient. The following day there was a moderate swelling of the lower lid, the cyst appearing porcelain gray. The lid was not painful. By August 7th, the swelling had disappeared and the cyst seemed to be obliterated. On August 10th, there was still some slight ecchymosis about the site of the needle puncture, but otherwise all signs of inflammation had cleared. On this date two minims of the same solution were injected into the cyst in the left eye, but the solution did not diffuse into the entire cyst because of the constrictions previously mentioned. The patient complained only of a slight stinging pain. There was very little reaction on the following day. On September 7th, that portion of the cyst which remained was injected with about one minim. At each injection there was some ecchymosis into the conjunctiva immediately following the penetration of the solution. On September 23rd, no trace of either cyst could be seen and photographs were made

of the conjunctiva (Fig. 3 and Fig. 4). On November 16th, one lobule of the cyst in the left eye seemed to be recurring. One month later this showed further filling and was injected again with 0.1 cc. of solution. It became chalky white after the injection and considerable hemorrhage occurred into the bulbar conjunctiva. On January 4, 1934, this cyst seemed entirely obliterated. This was almost six months after the first injection.

FIG. 3—(Weymann)—Right eye after injection (September 23).

FIG. 4—(Weymann)—Left eye after injection (September 23).

In short, the injection of one to two minims of full strength quinine urethane solution into these conjunctival cysts could be made with very little pain, the reaction was slight and subsided within a few days, and obliteration of the cysts followed the

injection where the solution came into contact with the entire cystic cavity. As was found by Kilbourne and Murray, repeated injections were necessary in some instances to completely obliterate the cystic cavity.

On September 19, 1933, an infant eleven weeks of age was seen with a capillary hemangioma four by five millimeters in size just opposite the inner canthus of the left eye. A second small hemangioma one and one-half millimeters in diameter lay just temporal to the larger one. On September 28th, 0.1 cc. of a dilution of one to three of a solution consisting of four parts quinine hydrochloride, two parts urethane, and thirty parts water was injected into the main tumor mass. Immediate blanching occurred. The following day there was some discoloration of the center of the tumor mass with less elevation of the entire tumor. This shrinking continued until October 14th, when it was noted that there was a mass giving a purplish shadow under the skin of the lower lid. This was interpreted as a continuation subcutaneously of the original growth. The mass on the surface of the skin also began to enlarge at this time so that after consultation with a dermatologist carbon dioxide snow was applied. Up until December 5th, five applications of carbon dioxide snow were made which seemed to have no effect in checking the growth of the mass in the lower lid. At this time 0.25 cc. of full strength of the above quinine urethane solution was injected into the middle of the growth in the lower lid. On the following day the lid was quite swollen with some ecchymosis. On December 26th, the tumor mass in the lower lid was somewhat smaller and 0.2 cc. of the same strength solution was injected into the cutaneous tumor at the inner canthus. After this, the lids became swollen with some ecchymosis and when this reaction had subsided the tumor mass seemed more sclerotic and had shown no further growth. Because Professor Elschnig was in Los Angeles on January 17th, he was asked to see the patient. Surgical removal of the hemangioma was advised by him. Accordingly, operation was done by Professor Elschnig at my request on January 23rd. At operation, there was very little bleeding for the tumor mass seemed to be fairly well demarcated by sclerotic tissue. Professor Elschnig was of the opinion that the sclerosing injections had simplified the operative procedure by making the outline of the tumor more definite. Healing was uneventful. On April 28, 1934, there was no evidence of recurrence of the growth.

Histological examination of the tumor showed it to be of the capillary type.

This patient had a congenital hemangioma of the capillary type which was exhibiting rapid growth and was uncontrolled by the application of carbon dioxide snow. The injection of quinine urethane solution apparently stopped the growth of the tumor, and in the quantities injected produced a moderate inflammatory reaction with ecchymosis but no sloughing. It is possible that if a metal ring had been used to stop the circulation in the growth when the injections were being made that better results might have been obtained. It was my opinion, and the opinion of Professor Elschnig at operation, that the sclerosis produced by the injections had made the outlines of the tumor more distinct through the formation of scar tissue and facilitated its removal.

A sclerosing agent for use in ophthalmic therapeutics should be capable of producing marked effect in small amounts and should have as little danger of producing sloughs as possible. This was the reason for my selection of the quinine urethane solution, along with the fact that it had previously been used for injection into the orbit by other writers without damage. It has the disadvantage of being toxic for those individuals who are sensitive to quinine, and of being so powerful that sloughing may occur if overdoses are used. In consideration of the possibility that a variococele of the orbit might sometime be encountered, it was decided to test the effect of injecting this solution into the orbits of rabbits. Also an attempt was made to cause the formation of conjunctival cysts in rabbits which might later be treated, but it was found impossible to produce such cysts by any of the methods tried.

As Kilbourne, Dodson, and Zeiler[7] had found that 0.5 cc. of quinine urethane solution could be injected into two kilogram rabbits without being fatal, six rabbits of a group of twelve of over this weight were injected with 0.5 cc. of standard strength quinine urethane solution in the left orbit. On the following day the rabbits showed marked subconjunctival hemorrhage and conjunctival edema. In two rabbits the injection was mostly subconjunctival, and in one of these, two days after injection, a keratitis occurred. Within two weeks after injection four of the six rabbits died, while all the controls were healthy. The dead animals were inadvertently destroyed by the caretaker, so that no cause of death was determined.

Accordingly, on June 6th, 0.2 cc. of quinine urethane was injected into the right orbit of six albino rabbits. The injection was made in the lower temporal quadrant. The following day one rabbit was found dead with a marked protrusion of the globe, and edema and hemorrhage into the conjunctiva. Grossly, the brain and intracranial portion of the optic nerve appeared normal. The globe showed marked petechial hemorrhages into the ciliary body. There was an infiltration of a polymorphonuclear type in the orbital fat with some edema. Two of the remaining five rabbits showed haziness of the cornea and congestion of the iris. All showed exophthalmos and ecchymosis of the conjunctiva.

Finally, one rabbit developed ulcerations of the cornea and another suffered with an active keratitis to the extent of complete functional destruction of the cornea. The third rabbit had a moderate degree of keratitis confined to the lower temporal quadrant. The fourth rabbit suffered a slough of the conjunctiva and orbital contents in the region of the injection, but this healed without damage to the globe. The fifth remaining rabbit never showed at any time any involvement of the globe and recovered with a normal-appearing eye.

The same five living albino rabbits who had the injection of full strength solution in the right eye, with the addition of one extra rabbit to make six, were injected into the left orbit with 0.2 cc. of a one-half dilution of the full strength solution used above. The following day all showed some irritation of the conjunctiva and slight proptosis. Two days later, only two rabbits showed any reaction, the eyes of the others appearing normal. These two showed some conjunctival swelling with engorgement of the iris vessels. One week later these eyes had also returned to normal, so that no permanent damage was caused to any eye by the intraorbital injection of the half strength solution.

The results of these experiments would tend to show that the injection of a full strength of the above-mentioned quinine urethane solution into the orbit is not to be undertaken lightly. Of course, one must consider that in the rabbit the amount of orbital tissue surrounding the eye is comparatively small, and that the sclera is much thinner than in the human eye. But of the living rabbits sixty per cent suffered permanent damage to the globe from an injection of only 0.2 cc. of full strength solution.

I should, therefore, certainly hesitate to inject 1.5 cc. of this solution into the human orbit, as is reported by Weekers.

From an analysis of his report, one sees that he injected 2 cc. of novocain prior to the injection of the quinine urethane solution. This, in my opinion, acted as a diluent for the quinine urethane solution and thus saved him from having such a serious inflammatory reaction that damage to the globe might have resulted.

From a survey of what has been written, and my own experimental clinical results, I would conclude that there is a definite but limited field in ophthalmic therapeutics for the use of sclerosing solutions as represented in this article by quinine and urethane. This solution should never be handled except with the utmost respect, and the patient should always be tested for his sensitivity to quinine before its use. The standard solution of four parts quinine hydrochloride, two parts urethane, and thirty parts water should in all cases be diluted from one to four times in making the first injections to determine the reactivity of the individual. Superficial neoplasms of a vascular type may be treated without fear of damage if no more than 0.2 cc. of solution is injected at any one time, and if sufficient interval is allowed between treatments to permit all reactions to subside. In treating such vascular growths, it is advisable to produce stasis of the circulation by means of a metal ring surrounding the growth. If later operative removal of the growth is necessary, this may be facilitated by the encapsulation caused by the injection. Obliteration of cysts or lymphoceles of the conjunctiva may be obtained by the injection of this solution, but here again it should first be tried in a diluted strength, and never more than 0.2 cc. injected at one time. Care should be exercised that the solution enters the cyst and not be injected subconjunctivally. If surgical removal of the cyst can be easily accomplished, I should advise that method of procedure rather than the injection.

The intraorbital injection of this solution into vascular tumors should be approached with the utmost caution. Undoubtedly the human orbital tissue will stand a fairly large dose without damage to the globe. But no harm will be accomplished if one first tries the smaller dose of 0.2 cc. with a one-half strength dilution. Then, according to the reaction produced, further injections of increasing strength may be made until obliteration of the tumor has been obtained. In those orbital tumors of a vascular nature, where no other method of treatment offers much chance of success with the conservation of vision, one is

certainly justified in trying the injection of quinine urethane as above outlined.

To sum up the whole situation, one might say that in the sclerosing solutions we have another method of treatment which, if properly handled, may prove of value in treating some conditions where no other method of treatment may be more easily accomplished. Where other and simpler therapeutic measures will suffice, the use of sclerosing solutions must be considered more dangerous to try, and not the method of choice.

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DISCUSSION

Dr. Sanford R. Gifford, Chicago: There are certainly occasions when it would be exceedingly convenient to have a safe means of obliterating vascular and cystic growths without resorting to surgery. The problem is to produce satisfactory obliteration of the offending structures without risk of damage to the cornea or other ocular tissues. I must admit having had no experience with the quinine solutions employed by Dr. Weymann, but was stimulated by his paper to find out more about these and other similar solutions.

In the case of cysts, while Dr. Weymann's result was highly satisfactory, a simpler and probably safer method seems to be one suggested by my father in 1923. He reported 7 cases of dermoid and the allied oil cysts of the orbit which were cured by incision and swabbing the epithelial lining thoroughly with pure trichloracetic acid. When such cysts are small and in the anterior part of the orbit excision is often perfectly simple and seems like a cleaner surgical procedure. When they are large, however, and penetrate deeply into the orbit or are tightly adherent to the periosteum, total removal may be impossible and destruction with trichloracetic acid is a perfectly effective and safe procedure.

To these cases of my father, I may add another which I feel could not have been attacked so successfully by any other means. A woman of sixty appeared at the clinic with a large mass in the inner angle of the left orbit. This had been present for many years, but had gradually increased in size until the globe was pushed temporally and forwards. There were no signs of inflammation. Lacrymation was present, but the lacrymal passages were freely permeable.

It was thought that the mass, presumably a dermoid, might be removed, but on incising the skin it proved to extend far back into the orbit. The wall was incised, permitting the escape of several drams of clear oily fluid. The inner wall was tightly fused with the periosteum, but there were no openings into the sinuses. The walls were dried thoroughly and swabbed several times with pure trichloracetic acid. The wound was closed and a pressure bandage applied. No aristol was introduced, as was done in some of my father's cases. Healing was uneventful and there has been no recurrence of the cyst. As in destruction of the lacrymal sac, the skin is painted with thick zinc oxide ointment to prevent any excess of acid from burning the skin, though if only a few drops at a time are used there should be no danger of this.

It has often been convenient to use this method in smaller retention cysts of the fornices, and when, in dissecting out a dermoid, part of the cyst is cut off leaving a stump containing epithelium, use of trichloracetic acid on this stump will often prevent a recurrence.

When dealing with vascular nævi or orbital varices, unfortunately, we are unable to employ this simple measure, as numerous large and small vascular spaces are present. The encapsulated hemangiomata of the orbit may be removed *in toto*, as a rule, but for the more diffuse nævi with many large vascular tributaries, this is often exceedingly difficult or im-

possible. Ligation is often difficult and ineffective. Radium will usually be employed where the vessels are small and near the skin and the results, with proper technique, are often exceedingly good. Where the vessels are larger and deeper, radon seeds may be implanted and left. The effect of these small implants, usually 2 mc., continues for three to four weeks and results in a type of sclerosis similar to, apparently, that produced by quinine solution. Each 2 mc. seed produces its destructive effect in a zone of 5 mm. about it, so by placing seeds 1 cm. apart a growth of considerable size may be destroyed with less damage to the overlying skin than when plaques or needles are applied from without. The reaction is less severe and more prolonged, and might be compared to that of a number of very minute injections carried out over the same period. The chief and a very important difference is that with radium we must always consider the possible danger to the crystalline lens. The skin also may be severely damaged by radium, especially as usually applied from without, and I have seen one complete coloboma of the lid result from its use. For these reasons, it is often preferable to employ diathermy-coagulation or electrolysis, where the location of the growth makes this possible.

On consulting with Dr. Geza de Takats, who conducts the Vascular Clinic at Northwestern University, I am told that the substance now preferred by him for injection of varicose veins is a solution of potassium oleate 5%. This has the advantage over the quinine solution and also over sodium morrhuate, which Dr. Weymann mentioned, that idiosyncrasy to it does not occur spontaneously nor is it produced by the repeated injections which are necessary in his work. It is also free from the systemic effects of quinine. This is used, of course, for injection into the vein. Dr. de Takats doubts if it would cause enough reaction and cicatrization when injected outside a vein, as must be done in orbital varices. We found that $\frac{3}{10}$ cc. of 10% solution injected subconjunctivally on ½ cc. behind the globe caused a quite severe reaction in rabbits, lasting two weeks. When smaller amounts of a 5% solution were used ($\frac{1}{10}$ cc. subconjunctivally and $\frac{3}{10}$ cc. retrobulbar) a moderate reaction occurred which subsided completely in a week. This left no scar tissue, apparently, and it seems that a fairly severe reaction is necessary to produce this when injecting outside the veins. It is this inability to inject into the vascular spaces accurately that will, for most of us, limit the indications for sclerosing solutions to a very occasional case. I have such a case now, a man with von Recklinghausen's disease, with tremendous hypertrophy of one side of his face and orbit, in whom I am going to try injections, because we have been taking out pieces of skin but still there is a great deal of hypertrophied tissue. We will be led, then, to prefer ligation or electro-coagulation when possible, with radon implants for cases with many deep diffuse vessels.

Dr. R. O. Rychener, Memphis: Dr. Weymann has taken a further step on the trail begun by Lebon, Santos Fernandez and Beauvois, who, as early as 1902, reported cures of cases of pulsating exophthalmos by the injection of the ophthalmic vein with 4% solution of gelatin. Since

then many other drugs and substances have been similarly used for a variety of pathological conditions. Of these, my personal experience has been limited to the use of absolute alcohol, which I have used with gratifying results in many cases of spastic entropion in the manner described by Elschnig, Dupuys-Dutemps and Wendell Hughes. I have also seen it used with but little effect in a capillary hemangioma of the brow, although Klinedinst in 1911 reported the cure of such a lesion of the lower eyelid by repeated injections of small doses of absolute alcohol.

The prospect of obtaining a safe solution for the injection of orbital angiomata is alluring. Some authorities contend that coagulating solutions should never be used without ligature and flatly condemn their use in the treatment of orbital angiomata. The experiences of Raverdino, Weekers and Weymann show that good results may be obtained by the judicious use of such solutions in guarded dosage, and undoubtedly more of us will be encouraged to attempt injection for orbital conditions, such as pulsating and intermittent exophthalmos, orbital varices and angiomata, in which other measures may fail.

I have had in my clinic care for the past eight years, a colored girl of twenty-six who has extreme exophthalmos of the left eye, which has ruined her entire life. She has been seen by practically every ophthalmologist in Memphis at one time or another. She has been operated twice, with confirmation of the diagnosis of inoperable hemangioma of the orbit and has been exposed to much roentgen therapy, which she now refuses, because of the terrific headaches which result after each treatment. In such a case, it seems to me that the use of a sclerosing solution would be justified, if not indicated.

Some of us have in our care cases of exophthalmos due to hemangiomata and, in some cases, due to pseudo-tumors, in which, in spite of operation procedure, radium, x-ray, we are unable to effect any cure.

It is likely that in a few such cases the injection of such solution as described by Dr. Weymann is indicated and justified.

Dr. Weymann is to be commended for his experimental work by which he has established the limits of a safe injection dosage of quinine and urethane.

DR. M. F. WEYMANN, closing: In response to Dr. Rychener, I might say that I do not wish to offer too much encouragement in the use of these solutions, for to me they are a method of last resort.

Concerning the potassium oleate mentioned by Dr. Gifford, it may be said that the reason for selecting quinine urethane in preference to the salts of fatty acids is that it has an anesthetic effect, whereas sodium morrhuate or potassium oleate is painful on injection, particularly outside the vessels.

One more patient was seen after this paper was written. This was a man who had a capillary hemangioma in the outer portion of the right lower lid. In this individual, hemostasis with a chalazion clamp was used while .1 cc. of one-half strength quinine urethane solution was injected into the tarsal growth.

There was no appearance of the hemangioma on the skin surface of the lid, but it could be seen on the margin from which point it extended back into the conjunctival surface. The patient desired its removal for cosmetic purposes because of the disfigurement caused by the discoloration on the lid margin. There was immediate rather severe pain after the injection of such an amount as to be surprising to me. This was probably due to the fact that the injection was made in the dense tarsal tissue. Following the injection there was a hypesthesia which lasted for about three weeks.

The first injection did not eliminate all of the growth so that a second injection was made a little over three weeks later. This second injection caused considerably less pain and probably would not have been painful at all had it been made one week following the first injection during the period of anesthesia. At the time of the second injection the chalazion clamp was left in place three minutes for the production of hemostasis and localization of the injected fluid.

Two days after the second injection there was very little edema of the lid remaining, but a slight slough occurred along the lower margin of the lid. After complete healing occurred practically the entire growth was obliterated but there was also a slight anatomical defect in the lid margin due to the slough. It is not noticeable and not sufficient to produce any functional damage. However, such a result is enough to cause a repetition of the precaution to start with low dilutions in the use of these solutions.

SUPPRESSION IN ANTIPATHY TO SINGLE VISION

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IT IS GENERALLY agreed that orthoptic training in children is of decided value. A recent survey by Guibor[1] establishes preference of orthoptic training over other approved methods either alone or combined. It even seems to have a place in alternating convergent squint although some question its value in this instance. Concerning stereopsis, Duane[2] states, "in some people it is absent or imperfect, in others it is moderately developed and in still others it is highly developed. In those in whom it is weak it can often be improved by practice; such practice serves as an incentive to the development and maintenance of binocular fixation." In adults, however, an entirely different problem is involved. If a defective fusion center is stimulated, symptoms of asthenopia may be invoked or greatly accentuated. The proof that binocular single vision can be produced from fusion training in individuals with suppression is still lacking. Peter[3] states, "in adults, however, the decision as to whether one shall allow suppression to continue or whether fusion training shall be pushed to its limit must be made after carefully weighing the evidence in each case as to which method is apt to yield the most comfort for the patient."

During the past few years there has been ample opportunity to study adult patients who have been more or less completely incapacitated by fusion training. The application of the principles used to produce suppression after such training and the treatment necessary in unsuccessful surgical cases indicates that suppression and not binocular single vision should at times be the aim of the ophthalmic surgeon. We believe that one can tell before treatment is begun whether binocular single vision may

be obtained from treatment or whether the inhibition, namely, suppression, should be allowed to continue. Furthermore, it seems probable that if binocular single vision is sufficiently developed, correction of the muscle imbalance alone will relieve symptoms without fusion training. At this time we are chiefly concerned with doubtful phorias and heterotropias of low degree, especially the correct analysis, the prognosis, and the procedure to follow.

BINOCULAR SINGLE VISION

Fusion and binocular single vision have to some extent been used as synonymous terms yet are quite different. Guibor[4] states that binocular single vision consists of second degree fusion according to Worth's classification of squint. Such fusion exists after the surgical correction of concomitant convergent squint or even in the presence of low degrees of constant squint.

> Mr. G. T., age 34, has had an alternating convergent strabismus (probably originally monocular) since two years of age. Treatment by correction of the refractive error without orthoptic training resulted in a very good cosmetic result. The refractive error at present is:
>
> | | | | | |
> |---|---|---|---|---|
> | R.E. | +1.00 S | +1.75 C | × 105 | 20/15 |
> | L.E. | +1.00 S | +1.00 C | × 45 | 20/15 |
>
> On screen test there is 5 D of convergent squint, which amount corresponds closely with the parallax test. Measurement of the deviation on the phorometer is impossible. Alternating macular suppression is present at all times. With the amblyoscope and stereoscope first and second degree fusion with some amplitude is present. On the Zeiss stereoscope and pfungstafel he locates correctly, 16 out of 18 objects. This would permit him to secure a private pilot's license, a hobby in which he is much interested. This stereopsis is present without fusion training in spite of a more or less constant squint of low degree.

In our opinion such fusion occurs in individuals who never have had and never will have binocular single vision. These persons have fusion when tested with the amblyoscope, stereoscope or other devices, yet instinctively suppress one image without the aid of prisms. It also occurs in individuals who have an antipathy to single vision, and should stereopsis be used as a test for the presence of binocular single vision it may bring much distress to both patient and physician.

In the majority of individuals, binocular single vision, together with stereopsis, develops involuntarily between the second month and the seventh year. The location of these functions is undoubtedly in the occipital cortex and as stated by Riley[5] an involuntary center is present in the occipital lobe for the purpose of fixed gaze. "Control by an occipital center over the maintenance of fixed gaze is a phenomenon for which a definite reason can be seen and for which there is considerable clinical evidence." Sherrington's law of reciprocal innervation showed the necessity for a subcortical center which was proved by Spiegel and Teschler[6] to exist in the vestibular nuclei. These nuclei distribute cortical impulses from the precentral gyrus and also from the occipital lobe. Furthermore, Riley[7] states that this occipital center may function after destruction of the cortical center when the eyes are rotated in the sphere of action of the paralyzed muscles for the preservation of fixation of the gaze. He terms it the oculogyric, while Spiegel calls it the optomotor center. According to Jackson's[8] views on mentation, lower centers such as the occipital "are the most organized, the least complex and the most automatic."

The presence of binocular fixation without binocular single vision was described by Maddox[9] nearly fifty years ago. The former is entirely objective, the latter subjective. The red glass test, bar reading, and four dots tests are those commonly used in the determination of binocular single vision. Bar reading, a subjective test, does not seem to us conclusive proof of binocular vision.

TESTS FOR BINOCULAR SINGLE VISION

If a red glass is held alternately before the right and left eye and the patient views a distant light, a pink color indicates fusion, red or white alone, suppression; both red and white signifying diplopia. According to Duane[10] the first indicates binocular vision, the last, binocular vision with diplopia. This cannot always be true, for correction of an imbalance with diplopia frequently results in suppression rather than binocular single vision. In adults, after fusion training, and in some individuals even before, it is possible with correction of the muscle imbalance to have one light superimposed on the other without fusion actually taking place. It is our belief that this is the condition which occurs in individuals with squint who have had some

fusion training, or in persons with defective but not suspended fusion.

The red glass test is admittedly not entirely satisfactory and it is especially difficult after fusion training in adults to tell whether suppression or fusion is the predominating factor, especially if the lights are superimposed. Attempts to develop a more satisfactory method have been futile. It is advisable to test binocular vision both for 6 and ⅓ meters. It is at times present at one distance and not at the other. When tested at ⅓ meter with an ophthalmoscopic lamp, the filament will appear distinctly red when binocular vision is present.

We know from a study of the neuromuscular mechanism of the eye that fusion is a subconscious effort or may become a conscious effort if developed by the stereoscope. Binocular vision would seem to be always subconscious. This contention is at least borne out by the fact that although stereopsis is often developed by training, we have never been able to develop satisfactory binocular vision when suppression has existed for a period of time.

DIPLOPIA

Diplopia should be considered a disorder of binocular projection, not of binocular vision. The correction of diplopia by prisms or surgery often results in suppression, not single vision. Diplopia results after the surgical correction of squint and disappears again only when suppression intervenes. Two most distressing examples of diplopia occurred in persons with squint following traumatic paralysis, but disappeared before muscular function had completely returned.

Diplopia is often present in low degrees of vertical deviation, especially of two or more degrees. Its correction, and particularly under-correction, results in alternating or monocular macula suppression and not binocular single vision as we had at first supposed. Slight over-correction of a vertical balance sometimes results in a vertical deviation in the opposite direction, at times with diplopia and more often with an increase of asthenopic symptoms. This may be the reason why vertical deviations have always been under-corrected, although we do not know the origin of such a rule.

SUPPRESSION

Suppression represents one state of defective fusion occurring especially in concomitant squint but it occurs occasionally in individuals who never had any deviations of the visual axes.

H. G. M., aged 23 years, has never had any deviation of the visual axes.

Refraction: R. E. —2.87 S
L. E. —3.50 S

Muscles: Distant. 1 D Exo. Near 4 D Exo. Red glass, alternating macula suppression. Stereoscope, good depth perception.

Suppression varies considerably under different conditions and is complete if present at all times. When the fusion is defective rather than absent, there is occasionally annoying diplopia. It may be peripheral or central (foveal suppression). Orthophoric and strabismic suppression vary considerably with the kind and amount of deviation. It is less well developed in hyperphoric deviations because they are essentially paralytic. In congenital paralysis of the external rectus, it is usually developed for the range of the paralyzed muscle and the individual so learns to accustom himself to this range that he is not disturbed with diplopia. It usually persists after the correction of concomitant convergent squint even when the two eyes fix together.

Suppression may be either alternating or monocular, usually depending upon the equality of vision and the state of refraction. In young children one can usually determine the state of the suppression by the type of squint present. It is usually partial in the intermittent type and particularly so in the periodic type, indicating that diplopia may be present to some extent until the squint becomes constant. Children of 4 or 5 years with a beginning concomitant squint often complain of diplopia. This subject will be more fully considered under the various types of squint.

ANTIPATHY TO SINGLE VISION

In certain individuals it is impossible to produce single vision with any correction of the muscle imbalance. Such cases were first described by von Graefe and designated as antipathy to single vision and as pointed out by Duane,[11] "seem to occur particularly in conjunction with incongruity."

Antipathy to single vision has been variously interpreted as inability to produce binocular single vision or inability to produce fusion with a stereoscope. It would appear to be an uncommon condition in concomitant esotropia, although Duane[12] describes its occurrence after the correction of this condition surgically. Undoubtedly it occurs more frequently in an exotropia and hypertropia where the correction of the imbalance fails to produce binocular vision and the addition of a slight over-correcting prism produces a considerable deviation in the opposite direction. When parallelism of the visual axes is obtained therapeutically there is either a marked increase in asthenopic symptoms or nature overcomes the parallelism and the deviation becomes manifest. This strabismus is paramount to suppression.

In individuals who have antipathy to single vision, the induction of suppression is the surest means for the relief of symptoms. Fusion training in such instances only increases the difficulty. On the other hand, Duane was of the opinion that in some cases there was a marked tendency toward fusion which it seemed possible to develop, thus relieving the antipathy and incongruity. It is our opinion, however, that macular suppression in adults can be more easily obtained in most cases with satisfactory relief and considerably less effort. It would appear, also, that the visual factor is quite as important as the muscular, and fusion would never be satisfactorily developed because of an inequality of the visual acuity.

METHODS FOR THE DETERMINATION OF ANTIPATHY TO SINGLE VISION

The muscle balance is determined with the aid of a phorometer, screen test, duction test and with the red glass as the most satisfactory test for binocular vision. *The exact correcting prisms for the imbalance found (preferably the rotary prisms in the phorometer) are then placed before the patient and the effect of such correction determined with the aid of a red glass. For the sake of convenience an instrument described by Herholsheimer has been found very convenient. It consists of a white plain lens with a red glass on one side, and a 2 diopter vertical prism and red glass are on the opposite side.* It saves much time in testing each eye separately for the presence of single vision. If binocular vision exists, fusion, of course, will take place. Dip-

lopia, suppression or superimposed images may also occur. If antipathy to single vision exists, it will be impossible to produce binocular single vision with any variation of the imbalance found. Should binocular vision or suppression be found on correcting the imbalance, it is well to find the range of correction which will produce the same result. In the case of E. E., suppression was produced with correction of esophoria, and the range was from 16 to 24 P.D. Also, one should not confuse superimposed images as a sign of true binocular vision. Patients can usually differentiate between the two as oftentimes one light overlaps the other and appears partially red and partially white.

F. R., construction engineer, requiring presbyopic correction at 46 for first time, had refractive error of:

R. E.	+ 1.25 S	+ .25 C	× 65
L. E.	+ 1.25 S	+ .25 C	× 115

Phorometer 3 D Exo. — 12 D Exo. Screen test, binocular fixation at 6 M., alternating divergent squint at ⅓ M., convergent near point 30 cm. monocular vision and when exophoria corrected and test with red glass, stated that the two lights were superimposed one upon the other.

Alger,[13] in 1926, presented some instances in which the suspension of binocular single vision was advisable. At this time he stressed the unimportance of binocular single vision in certain individuals and gave an excellent description of the practical principles involved. The means which he used for this suspension of single vision were: complete occlusion, the reduction of retinal stimulation and the use of drugs, such as atropin. He also mentioned the possible enucleation of one eye as an extreme necessity for the relief of asthenopia.

To his measures for the production of suppression may be added:

1. Correction of the imbalance with prisms — orthoptic suppression.
2. Correction of the imbalance by surgery — orthoptic suppression.
3. Increasing the deviation already present — strabismic suppression.

The value of this treatment is pointed out by Alger and others and substantiated by the fact that suppression is a dom-

inating influence of a higher cortical center over a subcortical or more automatic one. It is our belief that when the necessity for suppression arises, it would seem better to stimulate the inhibition than to exercise it with orthoptic training.

SUPPRESSION IN ESOTROPIA

Concomitant esotropia has its onset in childhood when suppression occurs early. Whether monocular or alternating, strabismic and orthophoric suppression are readily interchangeable and persist after surgical correction. Diplopia is seldom an annoying symptom after surgery. Certainly binocular fixation with alternating macular suppression approaches the ideal result and is enhanced by orthoptic training. Thus we have a definite reason for the widespread popularity of orthoptic training in convergent squint. It may also, in some cases, prevent the later development of a divergent squint following surgery.

In congenital paralysis of the external rectus, suppression is less complete than in the concomitant type and diplopia is avoided by adjustment of the head to the range of motion of the paralyzed eye. Orthoptic training would be contra-indicated in this situation according to the experience with the following case:

> A. L. M., aged 43, owner of an advertising agency, had had considerable asthenopia for the past 3 or 4 years. During that time he has been treating with an optometrist and oculist who have been giving him orthoptic exercises, because of a tendency of the right eye to turn in. From the patient's history, there was never any tendency to convergent squint before that time. When seen in May of this year, the asthenopia was so pronounced that it was impossible for him to do any work. He experienced considerable difficulty overcoming diplopia. The orthoptic exercises were increased about 2 years ago because his daughter, aged 4 years, developed a concomitant convergent squint of low degree and was treated by this method.
>
> Vision:
>
> | | | | | | |
> |---|---|---|---|---|---|
> | R.E. | 20/64 | — .50 S | — .75 C | × 180 | 20/20 |
> | L.E. | 20/25 | — .37 S | + .75 C | × 90 | 20/20 |
>
> Muscles: 12 D Eso. at 6 M; 8 D Eso. at ⅓ M. Rotation shows complete paralysis of the right external rectus of congenital origin; red glass shows diplopia which changes to suppression on increasing the correction of the left eye. He prefers to use the right eye for near and the left eye for

distant vision. Suppression also was easier by correcting 6 D Eso. Considerable improvement was noticed on discontinuing orthoptic exercises, using right eye for near, left eye for distance, although suppression is not easily maintained.

In adults, concomitant squint with suppression does not seem to respond to orthoptic training.

Miss E. E., aged 29, complains of muscle trouble; difficulty in focusing, headache and fatigue. She had alternating convergent squint as a child but does not think it has been manifest in late years. First had following prescription:

R.E.	+ .50 S	+ .25 C	× 180
L.E.	+ .25 S	+ .25 C	× 90

and later from an optometrist who prescribed: R.E., + 1.75 S — L.E., + 1.50 S, and gave her a course of muscle treatments for 3 months. There was a marked increase of symptoms following the orthoptic exercise. Refractive error under 2% homatropin was:

R.E.	+ 3.75 S	+ .25 C	× 50	20/25
L.E.	+ 3.50 S	+ .50 C	× 90	20/30

Muscle balance: 18 D Eso. at 6 M., 17 D Eso. at ⅓ M. Diplopia was present with the red glass. Suppression was present when esophoria was corrected. With correction of the refractive error the balance was 9 D Eso. at 6 M. and 8 D Exo. at ⅓ M. with the relief of symptoms for a time. Improvement continued for a year, at which time the muscle balance was 5 D Eso. at 6 M, 5 D Exo. at ⅓ M. In the summer of 1933, there was a recurrence of her symptoms. Her physical condition was good except for some nervousness. Muscle balance was 12 to 24 D Eso. at 6 M.; 10 D Exo. at ⅓ M. She has suppression when 16 to 24 D Eso. corrected, more than 24 D prism produced crossed diplopia, convergence not well sustained. Shortening of external rectus from 3 to 4 mm. was advised because of poor convergence.

SUPPRESSION IN EXOTROPIA

Divergence of the visual axes offers an entirely different problem from convergent. Long-standing deviations of high degree are associated with complete suppression and often equality of vision. Correction by surgical measures is quite as satisfactory as in convergent squint, resulting in orthophoric suppression if binocular fixation is obtained.

Exotropia of low degree is sometimes confused with exophoria, convergent insufficiency and divergence excess. It is readily distinguishable if the imbalance is corrected by prisms and then tested with the red glass. Stereopsis may be present even with antipathy to single vision. With the phorias there is fusion, while in tropias simultaneous or double vision exists. Alternating exotropias with binocular fixation and orthophoric suppression rarely exists. As stated by Peter,[14] "exotropia of low degree does not produce disfigurement or symptoms and its correction does not produce binocular vision." Individuals may have a convergence insufficiency for many years, and yet always maintain binocular single vision either at the expense of their nervous energy or accompanied by a few symptoms.

H. B. F., aged 53, revealed 12 to 16 D exophoria at 6 M.; 17 to 26 D Exo. at ⅓ M. For the past 15 to 20 years binocular single vision with occasional diplopia has always been present without any tendency toward disassociation between the two eyes. Comfort was fairly well maintained by the correction of 3 to 6 D prism, base in.

Also, W. S. M., aged 48, revealed 6 D Exo. at 6 M.; 20 D Exo. at ⅓ M. He had had considerable asthenopia but has constantly maintained binocular vision. Correction with 5 D prism, base in, divided between the two eyes gives considerable relief.

When such imbalance is corrected surgically in the presence of binocular single vision the corrected balance is maintained.

C. R. M., aged 29, a university student, complained of difficulty with close work, inability to hold a focus and marked asthenopia. He had been unable to read for several years.

Vision:	R.E.	—4.75 S	—.50 C	× 15	20/25
	L.E.	—5.25 S			20/25

Muscles: 6 D Exo. at 6 M.; 11 D Exo. at ⅓ M. Binocular vision was present. Prisms afforded but little relief. Operation: bilateral central tenotomy of the external recti; result: 2 D Exo. at 6 M.; 1 D Exo. at ⅓ M. Four years later his muscle balance was: orthophoria at 6 M; 3 D Exo. at ⅓ M. There has been complete relief of all symptoms.

On the other hand, the correction of convergence insufficiency in the absence of binocular vision increases the asthenopic symptoms already present.

Mr. K. H., aged 36, university professor, wished examination for refractive error. He complained of constant asthenopia.

Refractive error:

R.E.	—.25 S	+.62 C	× 40	20/20
L.E.	—.25 S	+.62 C	× 162	20/20

Muscle balance: 3 D Eso. at 6 M; 16 D Exo. at ⅓ M. Binocular vision was thought to be present because of superimposed images. In 1931, 1½ prism, base in, was incorporated in each eye. The glasses were never worn because of their discomfort. In 1933, muscle balance was unchanged. There was definite diplopia with the red glass. The diplopia increased with correction of the error. In 1933, the prisms were removed. The refractive error in the right eye was decreased. This permitted the right eye to be used for distance and the left eye for near. This correction enabled him to do unlimited reading with comfort for the first time in many years.

It should be stated further that because this type of imbalance develops in early adult life, suppression is not complete and particularly orthophoric suppression. The deviation is necessary for suppression and relief from asthenopia. If corrected by surgery a long period of discomfort ensues until nature again accomplishes divergence or diplopia makes it necessary to reoperate to reproduce the original deviation.

SUPPRESSION IN HYPERTROPIA

In the correction of hypertropia it is necessary to determine beforehand the result one expects to obtain if the imbalance is corrected. It is possible with a prismatic or surgical correction to obtain one of three things: (1) binocular vision, (2) suppression, or (3) a paradoxical hypertropia, when the original imbalance is over-corrected. Since the imbalance was originally paralytic in many cases, suppression is not as complete as in the convergent type. It is true, also, that hyperphoria sometimes complicates a convergent squint or is oftentimes actually the cause of it. If the correction of low degrees of hyperphoria alternating macular or monocular suppression sometimes results.

A. H. F., aged 51, complained of asthenopia, headache, and conjunctival irritation with occasional diplopia.

Refraction:

R.E.	+ .75 S	+.25 C	× 135	20/20
L.E.	+1.25 S	+.50 C	× 45	20/20

Muscle balance: 3 D Exo. and 3 D LH at 6 M.; 10 D Exo. —3 D LH at ⅓ M. Screen test, slight Exo. and binocular fixation. There was diplopia with the red glass and complete alternating suppression with the correction of 2 D of LH. Relief of symptoms was brought about with above correction.

L. W. F., aged 13, refraction:

R.E.	+8.00 S	20/25
L.E.	+8.00 S	20/40

Muscles: 2 D LH —3 D Exo. at 6 M.; 2 D LH —8 D Exo. at ⅓ M. Correcting 2 D LH produced complete suppression of the left eye with the red glass whereas previously diplopia had been present.

At first we believed that the correction of these low errors always resulted in binocular single vision and were much surprised to find suppression with a correction of 2 diopters of hyperphoria even when associated with diplopia. In the higher deviations of 5 to 8 or more diopters, the imbalance varies considerably from day to day and an exact correction of the imbalance is impossible. Orthophoric suppression does not usually result from the correction of the higher deviation. Under-correcting the total error is vitally important. This relieves the symptoms of diplopia, head tilting and asthenopia, while even the slightest over-correction will produce a paradoxical hyperphoria. In one instance, there was perfect balance after surgery which later resulted in 15 degrees of manifest hyperphoria following a correction after the occlusion test. As pointed out by Duane,[15] one should correct the diplopia and not the deviation. It is our opinion that suppression is much better obtained with a small amount of the original hyperphoria still present.

Miss E. T., school teacher, aged 29, had sought the counsel of many oculists for several years, and, in 1929, went to an optometrist, who gave her 25 weeks of muscle treatment with a rotating disc. Her refractive error was:

R.E.	—1.75 S	—3.75 C	×170	20/40
L.E.	+ .25 S	+1.25 C	×135	20/15

The patient, now a psychopath, states that her muscle balance was from 1 to 12 D Exo. and 4 to 8 D LH at 6 M.

She was able to fuse well with any instrument, but never had spontaneous diplopia. From 1930 to 1932, the following operations were performed by a very competent surgeon: tucking of the left inferior rectus, recession of the right superior rectus, and two central tenotomies of the right external rectus. When seen in March of this year there was a marked increase in symptoms, considerable loss of weight, and inability to adjust herself to surroundings. The muscle balance, which for a time was orthophoria, is now 3 to 5 D RH with lateral balance. There was marked inequality in size of the retinal images which cannot be corrected. Binocular single vision could not be produced with any correction of the imbalance, although simultaneous vision was momentarily present. Suppression was difficult to produce by any measure. She thinks she is much more uncomfortable with the present RH than the original LH. Although now under the care of a competent neuro-psychiatrist, suppression does not seem possible and the obsession of the RH is a disturbing factor which cannot be remedied in her present state of health. Solution of this problem may never be accomplished, and in this case the orthoptic training is considered the greatest evil.

In contrast to the above the following case under the care of one of us is presented:

Miss G. S., school teacher, aged 27, first seen in 1932 complaining of diplopia, marked head tilting and asthenopia. The vision was 20/20 in each eye.

Refraction:	R.E.		+.37 C	× 15
	L.E.	+.25 S	+.25 C	× 180

The muscle balance: 18 D to 30 D RH, 2 D to 4 D Exo. The right inferior rectus was paretic. In June, a 4½ mm. tucking of the right inferior rectus, and in July, 1932, a 2½ mm. recession of the right superior rectus was performed. The resulting error of 8 D RH was corrected by 6 D vertical prisms divided between the two eyes. There was considerable improvement in the head tilting and diplopia although the latter was present on looking down and an increase of prismatic correction to 10 D was necessary. In July, 1933, the measurements were as follows: with glasses: 1 to 10 D RH depending on position of head. Occlusion test for 24 hours — 6 D RH, and a definite LH with correction of more than 6 D. No glasses: 22 RH. Operation: Central tenotomy of left inferior rectus with immediate correction of diplopia and head tilting, with phorometer, a slight RH or LH which was variable. December, 1933, although dip-

lopia and head tilting were absent, asthenopia was at times marked. The phorometer showed 4 D LH, the red glass absent binocular single vision, and suppression produced with difficulty. In June, 1934, asthenopia and conjunctival irritation were annoying symptoms. There was about 2 D LH and suppression of the left eye with over-correction of the LH to:

+.50 S +.50 C × 180 1½ BD.

This suppression gave almost complete relief of symptoms. It is believed that orthoptic training for this patient was definitely contra-indicated.

The following cases are cited in an attempt to prove, first, when orthoptic training and surgery are indicated; and, secondly, when they are contra-indicated.

Case I

B. C., aged 9, first seen in June, 1933. Since the age of 3 years he had been treated for an occasional divergent squint which the parents thought remained unchanged. His vision was: right eye 20/20, left eye 20/20, and his refractive error was: +.37 S × 90 in each eye. Muscle balance: 28 D Exo. at 6 M.; 18 D Exo. at ⅓ M. There was no hyperphoria. The convergence was well sustained. Screen test showed about the same amount of deviation as with prisms. The stereoscope showed good depth perception and the red glass binocular single vision with correction of from 16 to 20 D Exo. Orthoptic training was continued for a period of six months at which time the left external rectus was recessed 4 mm. Immediate result was 16 to 20 D Exo. at 6 M.; which improved within 2 months to 4 D Exo. at 6 M., and orthophoria at ⅓ M. Binocular vision was definitely present.

The above case and the following four cases are examples of when orthoptic training and surgery are indicated.

Case II

W. L. G., married, housewife, aged 45, complains of photophobia and dull pain through eyes for a year. She has worn glasses since 7 years of age.

Vision:	R.E.	20/25	—.50 C	× 75	20/20
	L.E.	20/20	—.50 C	× 50	20/20

Muscles: 8 D Exo. at 6 M.; 8 D Exo. at ⅓ M. CNP = 18 cm. Binocular vision with correction of the exophoria.

Prescription:	R.E.	—.50 S	× 75	2½ D	PB 1
	L.E.	—.50 S	× 150	2½ C	PB 1

which gave a muscle balance of 5 D Exo. at 6 M. and ⅓ M. Operation advisable if pain persists.

Case III

W. H. O., aged 22, a clerk, complains of asthenopia and inability to do any sustained reading. Has worn glasses since 6 years of age.

Vision:	R.E.	6/100	—1.25 S	+ 2.25 C	× 180 20/25
	L.E.	18/100	—2.25 S	—1.25 C	× 180 20/25

Muscles: 12 D Eso. at 6 M.; D Eso. at ⅓ M. Patient has binocular vision with red glass over L.E. Diplopia with red glass over R.E.; while correcting Eso., gives binocular vision at all times.

Case IV

Mrs. L. A. B., female, aged 48 years. Complains of inability to read, carsickness in automobile and street-car.

Vision:	R.E.	20/25	+ .25 C	× 60	20/25
	L.E.	20/25	+ .50 C	× 90	20/25

Muscles: 7 D Exo. at 6 M.; 8 D Exo. at ⅓ M. Adduction 24 D, abduction 13 D. Divergence is demonstrated with screen test. Diplopia with red glass and binocular vision with 8 D prism, base in.

Case V

Mr. F. J., a barber by trade, complains of tired eyes and lids that smart and burn. He has worn glasses for the past year without relief of symptoms.

Vision:	R.E.	—3.00 S	—3.50 C	× 112	20/20
	L.E.	—1.00 S	+ 5.00 C	× 70	20/30

Muscles: 6 to 9 D Exo., 1 D RH at 6 M. and ⅓ M. Red glass double vision with marked tendency toward suppression of the right image. Fundi: vacuoles in right lens, and star-shaped opacity on posterior pole of left lens.

Prescription:	R.E.	Plano		
	L.E.	—1.00 S	+ 5.00 C	× 70 with + 1.00 add. for near work

The following cases are cited in an attempt to prove when there is preferential suspension of binocular single vision.

Case VI

V. N., aged 10, when first seen was complaining of sick headache and inability to do her schoolwork. There was periodic divergence of the right eye mostly at night. The vision was: R.E. 20/25 and L.E. 20/25, and the refractive error was:

R.E.	+ 1.25 S	+ .25 C	× 95
L.E.	+ 1.25 S	+ .12 C	× 85

Muscle balance was 24 D Exo., 4 D LH at 6 M.; 28 D Exo., 4 D LH at ⅓ M. Fusion on the stereoscope was equal to 2nd degree. With correction of the imbalance there was diplopia with the red glass and at times spontaneous diplopia. Prisms which almost corrected the crossed diplopia often produced a homonymous diplopia. Orthoptic training was practiced for 6 weeks, at which time the muscle balance was somewhat improved, but evidence of binocular vision could not be elicited. There was a definite antipathy to single vision so that orthoptic training was discontinued. A correction for the refractive error was prescribed and surgery, for the present at least, was considered not advisable. There has been considerable improvement since the correction of the refractive error.

Case VII

Mr. D. L. J., aged 38, complains of frontal headache.

R.E.	— .25 S	+ 1.00 C	× 90
L.E.	+ .75 S	+ 1.25 C	× 70

Above correction did not relieve symptoms and the left lens was changed to plano after 10 days. This also failed to give comfort, which was immediate after increasing the correction to:

L.E.	+ 2.00 S	+ 1.25 C	× 170

Case VIII

Mr. S. J., a white male, aged 19 years, and a student by occupation, complains of inability to do school work; photophobia and irritation of the lids. His glasses do not relieve his discomfort.

R.E.	20/20	+ .50 S	+ .37 C	× 90	20/20
L.E.	20/30	+ 1.25 S	+ .75 C	× 100	20/20

2% homatropin four times at 15 minute intervals. Muscles: 2 D Exo., 1½ LH at 6 M.; 8 D Exo., 1½ LH at ⅓ M.

Abduction 9 D, adduction 13 D. Screen test and phorometer correspond in amount. Red glass shows complete suppression at all times. The following prescription gave complete relief.

R.E.	+ .25 S	+ .37 C	× 90	20/20
L.E.	+ 1.25 S	+ .75 C	× 100	20/30

Case IX

Mrs. J. R. McL., married and a housewife, aged 34. She complains of headaches, carsickness, and is unable to do close work. Muscles: 6 D Exo. to 20 feet and 19 D at 15 inches. Abduction was 17 D and convergent nearpoint 7 inches. Adduction was unobtainable. With a 6 D prism base in there was diplopia with the red glass.

Cycloplegic refraction:

R.E.		— .75 C	× 45	20/20
L.E.	— 1.00 S	+ 1.25 C	× 180	20/20

The following prescription gave her immediate relief:

R.E.		+ .75 C	× 135
L.E.	— 1.00 S	+ 1.25 C	× 180

SUMMARY

1. The correction of doubtful phorias and tropias of low degree are analyzed from the standpoint of binocular single vision and suppression.

2. Concomitant convergent squint is corrected more satisfactorily because strabismic and orthophoric suppression are interchangeable.

3. Exotropia of low degree must at times remain uncorrected because the strabismus is paramount to suppression.

4. In hypertropia, correction of the diplopia and not the deviation should be the aim of the surgeon.

5. When single vision cannot be produced by correcting the imbalance, suppression gives more satisfactory results than orthoptic training.

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DISCUSSION

Dr. Luther C. Peter, Philadelphia: I believe we can all subscribe to the summary of the paper presented by Doctors Grant and Knapp. In our enthusiasm over the results which are now obtained by orthoptic training, one may become overzealous and forget to properly appraise the possibilities in each case. Harm can be done, even in children, by attempting to break up the habit of suppression in true alternating squint while nature has so ably provided for the comfort of such patients. In adult life, the decision to train or not to train must be made more frequently and, as the authors have pointed out, one should exercise the best of judgment as to when a "let alone" policy is indicated.

True alternating squint is exceedingly rare and when present suppression should not be disturbed. It is as truly normal for alternating squint as is fusion for the average individual. In the young group, however, it is my belief that sufficient care is not exercised to distinguish

between squint which is truly alternating and the accidentally alternating variety. The latter is curable, and the former hopelessly incurable insofar as fusion training is concerned.

In adults, one may be dealing with exactly the same varieties of the state of the fusion faculty, but if the case is one of weak fusion, whether it is the result of a pre-existing, accidentally alternating squint or one of weak fusion without the history of squint, our problem is a different one from that of childhood. The question should be asked, is the patient tolerably comfortable or is he symptomatically unhappy? If the latter, can he be made more comfortable by fusion training or will a "let alone" policy be the wiser course?

The answer to this latter query is postulated upon a number of possible factors. Did the patient acquire the power of suppression in childhood? Is the power of suppression better developed than fusion? Can fusion be strengthened in a given case or is it too poorly developed to respond? Is the refractive error the same in each eye or is there considerable difference in the size of the images? In many instances there is a sufficient degree of anisometropia present to give the appearance of weak fusion when the discomfort really is due to inability to properly fuse images which are too dissimilar to be fused into one. I believe this latter condition is more prevalent than is usually supposed and that dissimilarity in images is the important factor in many cases of apparent fusion discomfort.

This thought naturally leads one to consideration of the tests which the essayists suggest to determine "antipathy to single vision." The red glass admittedly is not a satisfactory test and conclusions drawn from it may be erroneous. The reasons for error are obvious. In order to fuse readily, a good fusion faculty is required as a pre-requisite, and, second, a perfectly coördinating motility is equally important to bring corresponding retinal points into proper relation for the exercise of fusion. A third factor, however, is similarity in the size, shape and color of the images to be fused. Difficulty may arise from either one of these components of perfect fusion and dissimilarity of images may not be the least of the three disturbing influences. If, therefore, one is to add to the dissimilarity by reducing the illumination by means of a red glass, and by furthermore changing the color of one image, there is an increasing disparity which may lead to a false conclusion both as to antipathy and as to fusion.

The red glass leads to some conclusion in that diplopia may be established, suppression may be present or the images may be fused. In the presence of dissimilarity in color, diplopia is easily evoked, even in low degrees of heterophoria, but this does not establish antipathy as an entity. It may simply mean that the fusion faculty is not strong. Under such circumstances the problem becomes one of ascertaining whether there is dissimilarity in the size of the images. A "let alone" policy is indicated if symptoms are not too pronounced. If symptoms are severe, an attempt should be made to overcome the difference.

If suppression is present in an adult, carried through from childhood, it is my belief that no attempt should be made to develop fusion. The average adult patient who can suppress is more comfortable than fusion exercises can make him. If, on the other hand, suppression has been acquired in adult life, it is imperfectly developed and fusion training is more likely to bring about comfort.

The entire matter is one of good judgment and the question of "to train or not to train" should be carefully weighed.

Finally, the phrase "antipathy to single vision" does not seem to me to convey the real state of existing conditions. It is, rather, a question of strength or weakness of fusion. If fusion is totally absent, there is neither antipathy to single vision nor is there a desire for single vision. The problem is solved by suppression. If fusion is weak, and dissimilarity in the size of the images can be excluded, each case becomes an individual problem which can only be solved satisfactorily by a careful weighing of the evidence.

While I cannot accept the term "antipathy to single vision" as expressive of the underlying conditions and cannot feel that the red glass test is much of a determining factor as to the best course to pursue in a given case, I believe that the conclusions drawn in this paper are timely and should receive our careful consideration.

Dr. John H. Dunnington, New York: I agree with the authors that in many cases suppression is more desirable than imperfect fusion. The enthusiasts are advocating stereoscopic exercises for every case with any impairment of fusion. This, I am sure, is a mistake. It is well to bear in mind that a patient with imperfect binocular vision is frequently greatly annoyed by it, while one with complete monocularity is seldom conscious of any defect. These facts have been clearly pointed out by Doctors Grant and Knapp, so I only wish to add my approval to their views that the treatment of these cases must be individual, and to agree with them in urging a most careful analysis of the case before instituting orthoptic training. I do not share with the authors their pessimistic opinion about exotropias of low degree. In my experience, these deviations have been just as satisfactorily corrected as have the esotropias. It seems to me that useful binocular single vision is obtained more often in this variety of squint than in the convergent type, and that if fusion is not secured suppression occurs just as readily after the removal of the squint. Postoperative diplopia has long been the *bête noir* of ophthalmologists, and rightly so, for there is nothing more distressing than the persistence of diplopia after the strabismus has been removed by operation. I do feel, however, that these unfortunate results will be reduced to a minimum if one is careful not to attempt fusion training in those cases where at best only an imperfect blending of the images can result. My practice has been to ignore the presence of the diplopia and, as a rule, suppression promptly develops. On the other hand, if by various exercises one keeps the patient constantly thinking of the other image, suppression is as difficult to secure as is binocular single vision.

DR. FRANK N. KNAPP, closing: We are very grateful to Dr. Peter and Dr. Dunnington for their discussions.

The question of the difference in the quality of vision was thoroughly considered in the paper, but insufficient time was had for its discussion.

I think that Dr. Dunnington has secured results in his exotropic cases because operation has been performed very early in life, in which type I think good results may be obtained. However, in the type which occurs much later in life, in the young adults and middle-aged people, the results which I have seen have not been so satisfactory.

DIVERGENCE PARALYSIS DUE TO HEAD INJURY

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BUFFALO

DIVERGENCE PARALYSIS is a pathological condition in the function of vision characterized by homonymous diplopia with separation of the double images practically the same in all meridians, greater for far vision than for near, the separation diminishing as the test object approaches the eyes and diplopia disappearing entirely at a near focus. With this clinical picture there is no limitation in the function of any one of the extra-ocular muscles. Covering either eye demonstrates the presence of convergence. Subjective symptoms of headache, confusion, dizziness and nausea are usually present early.

This condition as a clinical entity was first named and described by Parinaud[1] in 1883. He wrote subsequent articles on this subject and reported and discussed several later cases.[2-7] During the closing year of the last century Berry,[8] Duane,[9] Uhthoff,[10-11] Straub,[12] and others[13-23] reported cases and discussed the pathology and diagnosis.

In 1900, Bielschowsky[24] wrote upon and discussed this subject at length, reporting three cases, and since has published other papers[25-27] on this subject, his latest ideas appearing in his book,[28] published in 1932.

In 1901, Berry[29] in a comprehensive paper, which he read before the Ophthalmological Society of the United Kingdom, stated that he had seen a number of cases of which he reported one in detail.

In 1905, Duane[30] reported and discussed nine cases; some years later, Alger[31-32] also reported nine cases and in a personal communication tells me that he has seen several others since. Dunnington,[33] Dunphy,[34] and others[35-65] have reported cases and made valuable contributions to the subject. The recent article of Stokes[66] in the Archives, reporting five cases, is further indica-

tion that divergence paralysis — or so-called divergence paralysis, if you choose — is not a rare clinical picture.

This condition is now so well recognized that my only excuse for bringing it up here and attempting to add something of interest lies in the fact that of all the approximately sixty cases reported to date, only one has been attributed to injury. The causes usually given are epidemic encephalitis, multiple sclerosis, syphilis, pneumonia, lead poisoning, cardio-vascular disease and tumor of the brain. In 1901, Bielschowsky reported a case that followed, and was believed to be due to concussion of the brain; no other case has been reported related to injury. It has been my fortune to see four cases following injury and in three of them no other possible or probable cause was discovered.

Case No. 1 — H. T., male, age 24, carpenter. On January 6, 1921, he was struck on the top of his head by a block of wood which fell a distance of 6 meters. He sustained a scalp wound and fracture of the skull. He was taken to his home, where he regained consciousness a half hour later. Three weeks after his injury, he was seen by the writer, at which time he complained of headache, confusion and double vision. The double vision was homonymous and he described the separation of the images as being about 45 cm. at 6 meters, with no material variation of the separation of images in any direction of gaze. As the test light was brought toward him the separation of images gradually diminished and at 24 cm. from the eyes his diplopia disappeared. Physical, neurological, and laboratory examinations were all negative except x-ray of the skull, which demonstrated the presence of fracture in left parietal region, which by one attending surgeon was thought to be slightly depressed and operation was considered, but refused, and none was performed. He was under my observation for nearly a year, during which time the condition remained unchanged and his case was closed by the Industrial Commissioner with an award of compensation for loss of vision of one eye on the basis of loss of binocular vision. I saw him again three years later when the condition was still unchanged though he had returned to work and was less disturbed by his double vision. Again, in November of last year, the condition was still unchanged, though he is no longer troubled by double vision. At no time did he show any evidence of paresis of any of the ocular muscles. His lateral ocular movements, measured by the tropometer were, and still are, 48° in by 45° out for each eye.

Case No. 2 — J. H., male, age 20, electric lineman. December 10, 1931, he fell from a pole a distance of 6 meters, striking the top of his head on the ground, resulting in a depressed fracture of the skull in the right frontal region. He was hospitalized at once and operation performed to relieve pressure. Orbital hemorrhage caused considerable proptosis with infiltration of blood into the soft tissues of the lids and homonymous diplopia was present, due to impaired function of the right abducens. His physical examination was otherwise negative. Diplopia gradually disappeared with his general improvement and six weeks after the injury binocular single vision was present, but stereoscopic vision was defective. He returned to work three months after injury, and nine months later, one year after the injury, he again began to be disturbed by double vision which gradually increased to constant homonymous diplopia of some 15° to 20° for distance, separation of the images gradually lessening as the test light was brought toward him, and at 25 cm. his double vision disappeared. There was no material variation in the separation of the two images in any meridian. He was working and getting by with his occupation fairly well but did not attempt to drive a car on account of confused vision. Visual acuity was normal, as, also, were his fields of vision.

The one point in particular in this case which differs from that of Case No. 1 is that there was present immediately, or soon after his injury, homonymous diplopia due to impaired function of the right external rectus. Afterwards, diplopia disappeared but reappeared at a later date. Duane and others are authority for the statement that paralysis of divergence may be preceded by paresis of one or both abducens and several of the cases reported in the literature give such history. This case is still under observation and has not been disposed of by the Industrial Commissioner, though payment of the claim for disability on the basis of loss of binocular vision has been recommended.

Case No. 3 — F. J., male, age 30, steel mill worker. December 10, 1932, he was struck on top of his head by an iron gear cover which fell a distance of some 6 meters; he was rendered unconscious and sustained contused and lacerated wounds on top and on the left side of his head. He regained consciousness in a short time; he returned to his job and finished his day's work. He was then hospitalized for nine days, at the end of which time he appeared normal, with no complaints. X-ray failed to demonstrate the pres-

ence of fracture. He then returned to work and worked for about six weeks, when headaches and blurred vision developed. At about that time, eight weeks after his injury, he was seen by me for the first time. He had visual acuity of 20/30 in each eye with normal fields and no complaints of double vision. He had an esophoria of 6° at 6 meters, but a red glass over one eye produced homonymous diplopia, the same in all fields, and which disappeared as the test light approached the eyes. During the following few weeks his esophoria developed into an esotropia of about 20° with homonymous diplopia present and the character unchanged. The screen test demonstrated convergence. Physical laboratory and neurological examinations were all negative. His ocular rotations were normal in both eyes and neither eye showed any evidence of pathology. This man's condition has not improved. He is still greatly disturbed by his double vision and has not returned to work.

These three cases of divergence paralysis all followed severe head injuries and nothing else was found to which the condition might be attributed.

Case No. 4 — J. H., male, age 22, salesman, was seen by me through the courtesy of Dr. Thurber LeWin in April, 1933. His history was as follows: In August, 1931, while reaching into the back of an automobile, the rear cover of which was raised, the cover fell and struck him on the right side of his head and jammed the left side of his head against the body of the auto. He states that he was not rendered unconscious and was able to go on with his employment for the balance of that day and the following day. Then he complained so much of headache that he was hospitalized. He was examined by competent surgeons, internists, rhinologists and dentists, and fracture of the skull was demonstrated, through the left temporal bone. At the end of ten months he returned to work. During this entire period he had made no complaint of double vision. Six months later, about 16 months after his injury, he complained of confusion in his vision and was found to have 11° esophoria with part-time diplopia. The double vision gradually increased and became constant, always homonymous, and the separation of the images for distance about 12°, practically the same in all meridians. The screen test showed the presence of convergent deviation. Until about 16 months after injury he was able to drive an automobile without difficulty and complained of no confusion in his vision. His diplopia became constant and developed the characteristic picture of paralysis of divergence. Examination of the corneæ of both eyes showed, however, the presence of extensive, deep vas-

cularization characteristic of healed interstitial keratitis. His visual acuity was 20/30 and 20/50 improved to 20/25 in each eye with lenses correcting a considerable amount of astigmatism. His fields of vision were normal. Blood Wasserman was negative and was again negative after provocative treatment. Separation of images with the test light diminished as the light was brought toward the eyes and he had fusion at 25 cm. There was no limitation in the movements of either eye. Lateral rotations, measured with the tropometer, were 52° by 48°. His case fulfills all the requirements for diagnosis of paralysis of divergence, but the question arises as to whether it was due to his injury or to congenital lues.

The onset of the symptoms of divergent paralysis varied in all of these four cases. In Case No. 1, it was present when first seen, three weeks after injury. Its character never changed materially and it was permanent. In Case No. 2, paresis of the right external rectus appeared directly following an injury with fracture of the skull. Diplopia disappeared and symptoms of divergence paralysis did not appear until several months later. In Case No. 3, confusion and double vision began to appear some two months after head injury. In all three of these cases nothing else was discovered to which the condition could be attributed.

In the fourth case, that with the healed interstitial keratitis, the influence of syphilis cannot be disregarded. In this case, bearing in mind the ten months' disability following skull fracture with headache, vertigo, and a period of hospitalization directly following the injury, it seems justifiable to classify this along with the other three and especially so as intensive antiluetic treatment resulted in no relief from the symptoms. Furthermore, in the fifteen or more cases of divergence paralysis reported prior to 1900, no case was mentioned as being related to syphilis and in the approximately forty-five cases reported since 1900, seven have been attributed to syphilis, but none of these to the congenital form.

Nearly all of the medical cases which have been reported have been with sudden onset and, indeed, it is generally agreed that sudden onset is one of the characteristics of divergence paralysis. In these four traumatic cases reported, only the first presented the characteristic picture when first seen. In the other three, the onset has been gradual and the symptoms of confusion with transient diplopia have not, at first, been sufficient upon which to base an early diagnosis.

A considerable majority of the medical cases reported also have resulted in cure or disappearance of symptoms with the restoration of normal binocular vision, whereas in these four cases following injuries there has been no tendency to recovery in any one of them. In one, more than thirteen years have elapsed since the condition first made its appearance and the others have shown no signs of improvement after a period of some two or three years. In three of my four cases, fracture of the skull was demonstrated, and in the fourth, while not demonstrated, it cannot be ruled out.

The whole subject of paralysis of divergence and the condition with which it may sometimes be confused, spasm or excess of convergence, is closely related to the subject of fusion. There has been considerable argument relating to a fusion center or fusion faculty. A logical hypothesis for the presence of a fusion center has been frequently proposed, but no such point has ever been traced, and its presence, if such exists, is veiled in obscurity. The presence of a center for convergence is generally conceded and believed to lie in Perlia's nucleus, a group of central, unpaired nerve cells, lying in the midline between the right and left cell groups of oculo-motor nuclear system. The presence of a center for divergence is only hypothecated, but is urged by Straub(?), Bielschowsky, Duane, Dunnington and others, while Berry states that he has seen several cases of so-called divergence paralysis of which he reports only one in detail, but finally questions the correctness of the diagnosis of divergence paralysis inasmuch as it involves the admission of divergence enervation, which he regarded as problematical. Duke-Elder[67] disposes of the question of a center for divergence by saying that divergence can most usefully be regarded as a relaxation of convergence. In a number of cases reported and described, some confusion exists as to a positive differentiation between paralysis of divergence and spasm of convergence, but the differentiation should not be difficult if we accept the dictum of Duane that the separation of the two images tends to lessen in a definite ratio as the object observed at a distant point approaches the observing eye.

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DISCUSSION

Dr. E. B. Dunphy, Boston: Most ophthalmologists now believe in the existence of an active divergence factor in the complex mechanism controlling ocular parallelism. Because a center for divergence has never been demonstrated anatomically, some claim that the clinical entity known as divergence paralysis is merely a spasm of convergence. There is something to be said for this point of view. For instance, when complete muscular relaxation takes place during sleep, it is a well-known fact that the eyes usually assume a position of divergence. Therefore, a constant contraction of both internal recti muscles must be required to keep the visual axes parallel. According to this theory, then, divergence is a purely passive function and is merely the result of a relaxation of convergence.

But this hypothesis cannot explain certain cases reported by Parinaud and by Duane in which the divergence paralysis was complicated by a paralysis of convergence. The hypothesis clearly contradicts itself here, for the divergence paralysis cannot be caused by a spasm of convergence when the convergence is already paralyzed.

Furthermore, as Duane has pointed out, it is contrary to physiological custom to ascribe both convergence and divergence to the contraction and relaxation of the internal recti alone. Elsewhere in the body, a joint is not flexed by one group of muscles and then unflexed simply by relaxation of the same muscles. Antagonistic muscle groups always participate. Adler has shown that the law of reciprocal innervation applies to the conjugate movements of the extraocular muscles in man, and it would seem logical that the disjunctive movements of convergence and divergence must be governed by the same principles.

I have seen only three cases of divergence paralysis and none was due to trauma.

In Dr. Weed's cases I am impressed by the period of time elapsing between the injury and the occurrence of the divergence paralysis. In the first case it was three weeks. In the second case it was one year, barring the short period of diplopia immediately following the accident, which might well be attributed to the pressure from an orbital hemorrhage. In the third case it was two months, and in the last case it was not until sixteen months after injury that the patient complained of diplopia.

I would like to ask Dr. Weed if he can explain these varying periods of time before the divergence paralysis set in. One would naturally think, if the trauma was the cause, that the diplopia would appear within two or three days after the head injury. Perhaps it might be explained on the assumption of a tiny hemorrhage somewhere near the hypothetical location of the divergence center. Organization of this hemorrhage with resulting scar formation might have impinged upon the center, causing the paralysis.

It is interesting to speculate upon the existence of a divergence center, but until someone is fortunate enough to obtain an autopsy on a case of divergence paralysis, the exact location of the center will remain in doubt.

Dr. F. H. Adler, Philadelphia: I think everyone who reviews these cases of divergence paralysis must be struck by one main feature and that is that in a very high proportion of them, sooner or later, there develops a paralysis of one or both of the external recti muscles.

If one compares the situation in cases of convergence paralysis seen so often after epidemic encephalitis, you are struck immediately by the fact that we do not see paralysis of the internal recti in these cases.

Further, the existence of a divergent center has not been shown either histologically or physiologically, whereas the reverse is the case for the center of convergence, namely, Perlia's nucleus, which Dr. Weed mentioned. The fact should, it seems to me, make one immediately suspicious that we may not be dealing with the kind of an entity which the definition or diagnosis implies, namely, we may not be dealing with a paralysis of a supernuclear cell.

It is thinkable (and I merely make this as a suggestion) that we may be dealing in some of these cases, at least, with an early paresis of one of the external rectus muscles. We know that if the external rectus muscle on one side is paralyzed completely, the eye can still turn out from a position of adduction to the midline by virtue of relaxation to the internal rectus. In an attempt to go beyond the midline, if the external rectus is not fully paralyzed, its action is aided by the superior and inferior obliques.

I am impressed by the fact that in Case 1, which Dr. Weed mentions, and in Case 3, the rotations as measured by the tropometer were, in Case 1, 48 degrees in and 45 degrees out in each case — 45 degrees I do not believe to be a full rotation outward — and in Case 3 it was 52 degrees in and 48 degrees out. The rotation, therefore, in these cases, outward was not, I believe, the physiological limit.

In Case 2, Dr. Weed mentions the fact that the patient did have a definite paralysis of the right external rectus, and the diplopia disappeared after a while, to recur later on.

It is also evident, I believe, that Dr. Weed feels that some of the cases reported in the literature may fall under this heading, for he makes the statement that in the case reported by Bielschowsky, the wearing of 3 degree prisms, base out, before each eye resulted in the permanent relief of the diplopia, and this leaves no doubt as to the correctness of the diagnosis, unless we may classify it as a partial paralysis.

Therefore, I believe that we should be very careful before committing ourselves to a diagnosis of this entity without more careful evidence. It is still possible that we are dealing with early and partial paresis of an external rectus muscle.

Dr. Harry M. Weed, closing: I greatly appreciate the discussions of Dr. Dunphy and Dr. Adler.

I have no explanation of the delayed symptoms. The clinical picture of divergence paralysis presents a group of signs and symptoms that are fairly well recognized and probably will undergo little change, but exactly what takes place in the brain to produce this clinical picture is not well

understood, and is it quite possible that a different terminology may be applied.

As for paralysis of one of the external recti appearing later, that too should become better understood as more is learned about the pathology. In my first case, now of thirteen years' duration, the tropometer shows exactly the same ocular rotations in both eyes as at the time of, or immediately following, the injury.

The direct ratio of Duane's, which he mentions as representing the lessening of the separation of images as they approach the eye, is merely the converse of the ratio for paralysis of convergence. In paralysis of divergence he states as a rather definite law that as the test object approaches the eye the esotropia at 1 m. is 3° less than for distance, at ½ m. 7° less, and at ¼ m. 15° less. Therefore, in a case of esotropia of 15° at 6 m. one would expect binocular single vision at about ¼ of a meter, or 25 cm. This ratio does not apply to spasms of convergence and, therefore, may be used as an aid in differential diagnosis.

An interesting, if not pertinent, commentary is that the subject of divergence paralysis is treated in the text of the old Fuchs-Duane, and Duane also comments on it in his footnotes, whereas in the recent edition of Fuchs-Salzmann-Brown I can find no mention whatever of the subject.

DETACHMENT OF THE INTERNAL LIMITING MEMBRANE OF THE RETINA

(HYALOID MEMBRANE)*

DONALD J. LYLE, M.D.

CINCINNATI

DETACHMENT of the internal limiting membrane of the retina, a glass membrane of one to two microns in thickness that lies between the retina and the vitreous, is not of infrequent occurrence. It is seen in nearly all cases of so-called proliferating retinitis where many of the bands or bundles usually described as connective tissue fibers are actually membrane detachments (Fig. 1). This detachment is also found quite frequently in chorio-retinitis (Fig. 2), and in diseases involving the vitreous (Fig. 3).

Whenever there is an area of retinitis, or chorio-retinitis, scar tissue is found as a sequel to the inflammatory process. The retraction of this connective tissue, which has bound the internal limiting membrane to the retina, exerts a pull upon the membrane, stretching it, so that it separates from the concave surface of the retina (Fig. 4). Thus, a detachment, usually narrow, is formed spanning the retina from the point of the lesion to another place on the arc of the retina, where it is firmly adherent to the underlying tissue. This other site of attachment is often

* A difference of opinion exists as to the presence or absence of a membrane between the retina and vitreous. If such a membrane is present there is disagreement as to its exact derivation. Briefly, there are four views:

1. No membrane exists between the retina and vitreous. (Mawas & Magitot, 1912) (Magitot, 1910) (Magitot & Mawas, 1913).
2. A hyloid membrane is definitely present. (Dejean, 1926)
   Such a membrane is present only in the morbid state. (Pillat, 1922) (Kraupa, 1923) (Isakowitzi, 1926).
3. There is no actual hyaloid membrane, but a definite thickening or condensation of the peripheral vitreous, as seen on surface colloids, is present. (Duke-Elder, 1929) (Mann, 1928).
4. A definite internal limiting membrane of the retina exists. (Mann, 1928) (Duke-Elder, 1929).

The author accepts the opinion expressed by Salzmann (1912) in his statement that there is anatomically one membrane separating the vitreous from the retina.

FIG. 1 — (Lyle) — Proliferating retinitis. The white bands radiating from the papilla to the upper and lower right margin of the photograph, passing over retinal vessels with relatively normal retina in the background, are detachments of the internal limiting membrane of the retina. The left half of the fundus shows a retinal detachment. These two types of detachment are separated by a hazy band extending across the picture lying forward in the vitreous, having penetrated the internal limiting membrane.

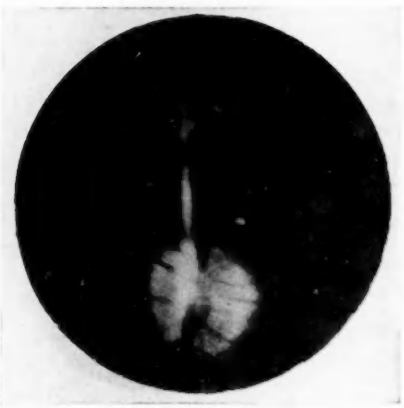

FIG. 2 — (Lyle) — A detachment of the internal limiting membrane of the retina extending from an old retinal lesion to the papilla.

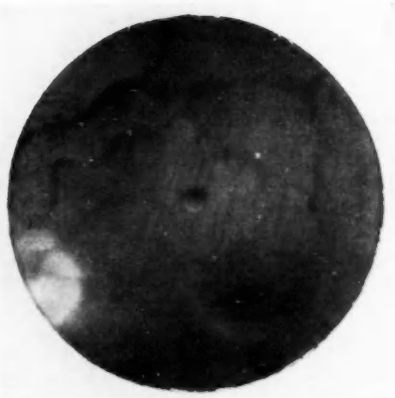

FIG. 3 — (Lyle) — Shrinkage of the vitreous with beginning separation of the internal limiting membrane of the retina in a broad area composed of a series of membrane folds extending from the macula upward to a retinal vessel. The tension of this eye is very low. Fine dust-like opacities fill the vitreous, which accounts for the indistinct photograph.

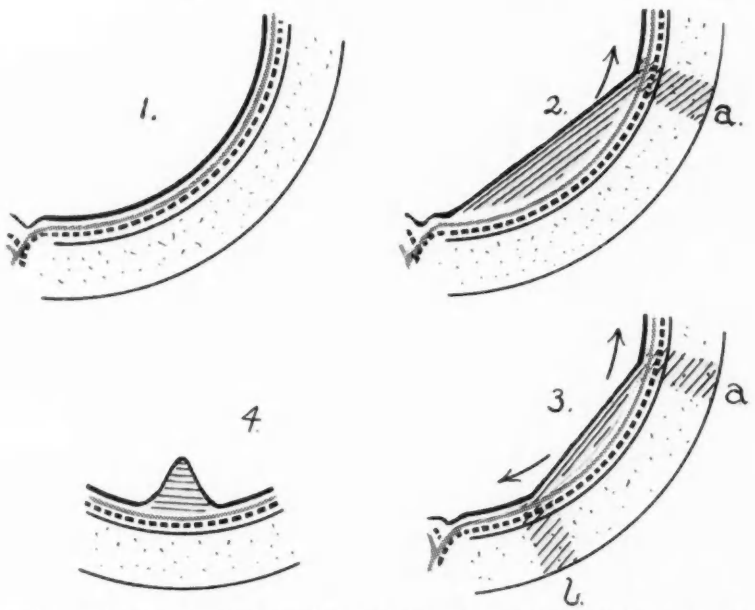

FIG. 4 — (Lyle) — Schematic drawings of detachment of internal limiting membrane of the retina. (1) Normal fundus. (2) Detachment of internal limiting membrane. Primary lesion is at '*a*'. Detachment extends to the papilla. (3) Detachment of internal limiting membrane. Lesions are at '*a*' and '*b*' with the detachment spanning the normal fundus between. Arrows indicate direction of pull. (4) Cross-section of the detachment of the internal limiting membrane.

the papilla (Figs. 5, 6, 7), though it may be the ora serrata, to both of which places the internal limiting membrane is normally very adherent. Occasionally, the span may either stretch between two separate retinal lesions or radiate from one central point. Shrinkage of the vitreous results in membrane separation by tearing it from its normal attachment to the retina. Gonin (1928) describes spontaneous retinal detachments due to tearing of retinal tissue caused by dragging of the vitreous.

In order to determine the relations of the internal limiting membrane of the retina and vitreous, which are such that slight retraction of the retina or shrinkage of the vitreous separates the membrane from its retinal attachments, it is necessary to study the developmental stages of these parts.

The supporting framework of the retina consists, for the most part, of fibers which have developed from the round cells of Müller and which ramify throughout all the layers of the retina. In embryological development, Müller cells are first seen in the primitive or proliferating layer of the inner wall of the optic cup (Mann, 1928). These cells migrate inward as the

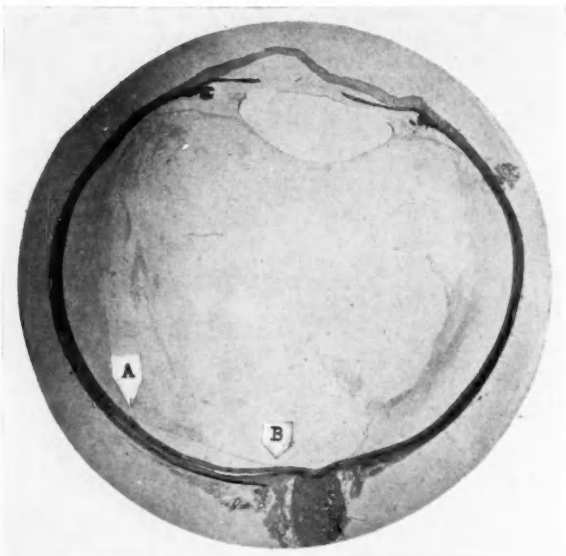

FIG. 5 — (Lyle) — A section of an eye with preoperative diagnosis of hemorrhagic retinitis. The internal limiting membrane is shown detached between a circumscribed retinal lesion at *'A'* and the papilla *'B'*.

retina develops and are found in the inner neuroglial layer, which finally contributes to the inner nuclear layer of the retina. Migrating still further, a few cells are next seen in the ganglion cell layer. Processes of these Müller cells extend inward to form the basal cones or foot-plates of the inner retinal surface and finally coalesce to form the internal limiting membrane of the retina.

Though the origin of the vitreous body is not firmly established* and is the subject of much discussion among embryologists, there is little doubt but that retinal elements penetrate into the optic cup. These processes join with others from the surface ectoderm, and possibly with mesodermal invading cells, to form a framework upon which and around which the vitreous

* The derivation of the vitreous has been attributed to the following primitive tissues:

1. Ectoderm — neural. (Wolfrum, 1907) (Mawas and Magitot, 1912).
2. Ectoderm — surface (lenticular). (v. Lenhossek, 1903) (Beckwith, 1927).
3. Ectoderm — both neural and surface (lenticular). (Cirincione, 1904). (Duke-Elder, 1933).
4. Mesoderm. (Dejean, 1923-4) (Monesi, 1926).
5. Ectoderm and Mesoderm. (Howard, 1920) (Druault, 1913) (von Szily, 1908) (Mann, 1927).

FIG. 6 — (Lyle) — A higher magnification of area at 'A' (Fig. 5) showing a retinal lesion the retraction of which has caused a membrane detachment.

FIG. 7 — (Lyle) — The papillary end of the membrane detachment shown at 'B' (Fig. 5).

develops. Even though, at later stages, the formation of the foot-plates and the limiting membrane separates the vitreous from the retina proper, these fibers from the retina and from the vitreous remain in permanent attachment to the internal limiting membrane. Later, mesodermal tissue may enter into the formation of the vitreous, but with this we are not concerned in this discussion.

The argument the author desires to develop is that when retraction of the retina, which is adherent to the membrane, or when shrinkage of the vitreous, which is also adherent to the membrane, occurs this membrane separates from its retinal attachments and remains attached to the vitreous body. It forms, first, a loose detachment; later, a taut bridge spanning the retinal arc between points where the membrane is more firmly attached. Still later, the membrane may entirely separate excepting at the papilla and ora serrata in an infundibular detachment.

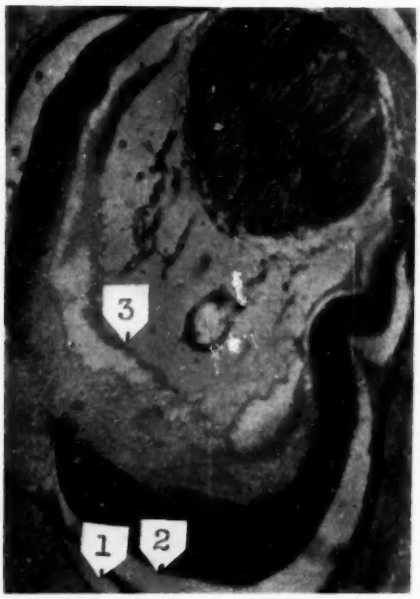

FIG. 8 — (Lyle) — Section through the eye of a 30 mm. human embryo. (1) Space between the choroidal mesoderm and the outer layer of the optic cup (pigment epithelium). (2) Space between the pigment epithelium and the inner wall of the optic cup (primitive retina). (3) Space between the primitive retina and the developing internal limiting membrane of the retina.

A section through the eye of a thirty millimeter human embryo (Fig. 8) shows spaces in the developing eye of this stage which, as potential spaces in the adult eye, are the locations of planes of separation or detachment due to various causes. These spaces are:

1. Between the pigment epithelium (outer wall of optic cup) and the choroidal mesoderm.
2. Between the pigment epithelium and the primitive retina (inner wall of the optic cup).
3. Between the marginal layer of the primitive retina and the developing internal limiting membrane of the retina (foot-plates of processes from Müller cells).

In the adult eye a separation, as described in the first instances, indicates a detachment of the retina including the pig-

ment layer. In the second instance, the retina would be detached excepting the pigment layer, which remains in contact with the choroid in its usual relation (the usual clinical detachment). In the third instance, there would be a detachment of the internal limiting membrane from the retina proper. It is not infrequently noted, in the study of sections of adult eye specimens where there has been no detachment or separation of the retina or the limiting membrane upon observation in the living eye with the ophthalmoscope, and when the eye has been normal in other respects, that a separation occurs in these before-mentioned places frequently because of distortion or shrinkage in laboratory preparation. This indicates the delicacy of adherence between these layers of tissue.

The chief causes of traction upon the internal limiting membrane of the retina leading to its detachment are those lesions the results of which induce scar formation with adhesions and contractions through inflammations or, hemorrhages in, and injuries to the retina, and shrinkage of the vitreous from any cause.

In the developing eye the vitreous is very closely associated with the papilla and ora serrata and generally less firmly connected with the internal limiting membrane. Where the vitreous is in proximity to the retina it is thickened into a posterior border layer which consists of a denser layer of fine striations parallel with the surface from which occasionally fibrilla are given off into the vitreous. Duke-Elder (1929) believes this border layer to be merely of the nature of a surface condensation as almost invariably occurs on the surface of a colloid. Whatever its nature, the attachment of the vitreous to the internal limiting membrane seems quite secure, as in the separation of the vitreous from its approximation to the retina, through any cause, the internal limiting membrane remains adherent to the posterior border layer and is detached from the retina. The vitreous is, however, loosely connected with the retina proper by fibrillar processes relayed through, or directly passing through, the internal limiting membrane. Hughes (1929) noted parts of the retina usually adhered to the vitreous after its removal from the globe, but these pieces could be easily brushed off with a soft camel-hair brush. Clinically, there appears to be but one membrane lying between and associated with both retina and vitreous which has developed as a product of the retina or vitreous or both. This

membrane, if retinal only, when detached, at least, carries with it the closely packed elements of the posterior border layer of the vitreous which, with the folds of the membrane, increases its thickness and aids its visibility when viewed in section or with the ophthalmoscope.

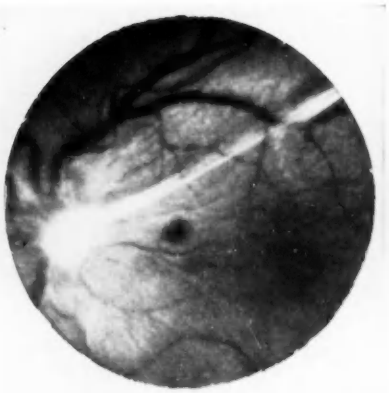

FIG. 9 — (Lyle) — A typical detachment of the internal limiting membrane of the retina which extends from a small circumscribed lesion in the periphery of the retina to the papilla. Note that it passes over the smaller retinal vessels, but is slightly influenced by a more superficial larger vessel.

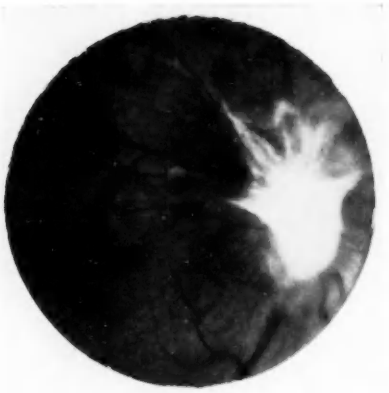

FIG. 10 — (Lyle) — Proliferating retinitis results often in contraction of the newly-formed tissue resulting in stretching of the internal limiting membrane over large areas, as is seen in the lines of tension radiating from the mass lying above the papilla.

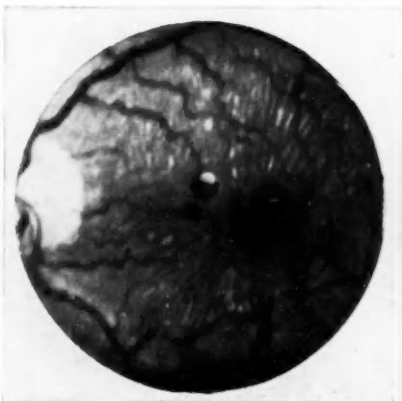

FIG. 11 — (Lyle) — Refractile lines or lines of tension of the internal limiting membrane are seen in a macular star formation which remains after the subsidence of a renal retinitis.

FIG. 12 — (Lyle) — A rupture of the choroid and retina in the macular area with scar formation and radiating lines of tension resulting in a slight separation of the internal limiting membrane over a broad area.

The diagnostic characteristics of a detachment of the internal limiting membrane of the retina (Fig. 9) are:

1. A narrow (until advanced stage) white fold, frequently single, occasionally several folds coursing parallel, sometimes stellate radiating from a point or small circumscribed lesion.
2. After separation it becomes elevated from the retina, greatest, of course, at the center of the span.
3. It arises at a retinal or neuro-retinal lesion unless caused by shrinkage of the vitreous when the retina may show no lesion. In this latter case the detachment is usually broader.
4. It terminates where the membrane is normally more securely attached as at the papilla, ora serrata, or at another retinal lesion. Sometimes a large vessel or apparently normal retina temporarily arrests the separation.

It is the author's belief that proliferating retinitis usually originates with an exudative retinitis, chorio-retinitis or neuro-retinitis, with or without hemorrhage, and that the sequelæ are, among others, exudates which produce connective tissue with the property of contraction. It is this contraction that causes a detachment of the internal limiting membrane. If the process extends to deeper layers, the entire retina is often detached (Fig. 10).

Moore (1922) refers to the star figure seen in the macular areas of renal retinitis caused by exudates. He states, "In some cases, previous to the development of the star, a complete figure of very fine lines may be seen radiating all around the yellow spot as a center. These lines have the appearance of refractile lines or lines of tension and suggest a sort of scaffolding around which star figures may be laid down." The photograph presented (Fig. 11) shows the fundus of a case of renal retinitis after the absorption of the stellate exudates and the cessation of the swelling. These "lines of tension" are due, no doubt, to a radial folding or detachment of the internal limiting membrane of the retina, as the edema and exudates have disappeared. Anderson (1931) describes a somewhat similar picture, finding "star-like" radiations from the macula after injuries, irido-cyclitis and

keratitis. He attributes this formation to folds of the internal limiting membrane at the macula. A photograph of a fundus of this description, caused by a rupture of the retina and choroid in the macula is presented here (Fig. 12). This rupture is surrounded by pigment and radiating folds of the internal limiting membrane.

Frequently, in papillitis and papilledema, during the acute stage or time of greatest swelling, one notices linear striæ radiating from the disc, macula or elsewhere in the paramacular area. This condition has been attributed to cellular exudation or edema of the nerve fiber layer of the retina. If, at the termination of the acute stage, these striæ disappear, these causes were probably the chief factors. If, on the other hand, as in the photograph here presented (Fig. 13), the striæ persist and, in fact, become more prominent after the recession of the swelling and inflammation, they are probably due to the folding of the internal limiting membrane. Later, through atrophy of the nerve fiber layer, light reflexes, much more marked than normal, appear along one side of the larger blood vessels. These reflexes are caused by the stretched internal membrane between the vessel and receding retina (Fig. 14). These streaks must not be confused with perivasculitis, in which the lymph spaces on both sides of the vessel are visible.

On the other hand, swelling of the nerve fiber layer will often throw the internal limiting membrane into folds not necessarily coursing parallel with the nerve fibers (Fig. 15). Indeed, quite often the serrated undulations are at right angles to the direction of the nerve fibers, apparently being stretched between retinal vessels or places of firmer contact.

Normal retinal light reflexes occurring most often in the macular and paramacular areas of hypermetropic and emmetropic eyes of juveniles are reflections from the internal limiting membrane of the retina. Retinal lesions may distort these reflexes throwing them into tension lines by contraction of the healing areas (Fig. 16).

Detachment of the internal limiting membrane of the retina should be differentiated from the following conditions which it most resembles or with which it is most easily confused.

Detachment of the retina as compared with detachment of its internal membrane is usually broader, with oval, round, or wavy border. The retinal blood vessels ride upon the surface

of the retinal detachment. The site of the retinal detachment may be found anywhere in the fundus, though it is seldom seen to radiate from the papilla, as so often occurs in detachment of the internal limiting membrane.

FIG. 13 — (Lyle) — Optic atrophy following optic neuritis. The internal limiting membrane is thrown into narrow folds as is seen by the striate light reflexes between the papilla and macula.

FIG. 14 — (Lyle) — Light streaks along one side of retinal vessels indicative of shrinkage (atrophy) of the nerve fiber layer which stretches the internal limiting membrane of the retina between the vessel and the receding retina. Occasionally a similar but less marked streak is seen in the normal eye.

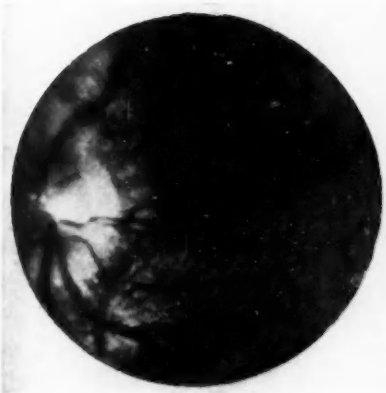

FIG. 15 — (Lyle) — A choked disc due to a cerebellar tumor. The parallel coursing white striæ are due to an edema of the papilla and nerve fiber layer with tension on the internal limiting membrane resulting in grooves which are seen to pass over the smaller retinal vessels, but are stopped by the larger ones.

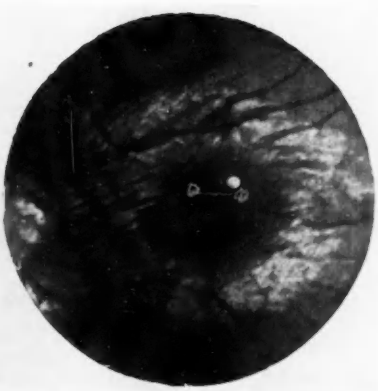

FIG. 16 — (Lyle) — A normal retinal reflex thrown into lines of tension because of a nearby circumscribed retinal lesion which through contraction has involved the internal limiting membrane.

Linear markings of the fundus, usually white, are seen in striate choroiditis, choroidal sclerosis and after reattachment of the retina. The courses of these lines are variable and not straight, as is seen in membrane detachments where tension plays a part. These striæ also lie behind the retinal vessels.

Medullated nerve fibers radiating from the papilla may be confused with detachment of the internal limiting membrane of the retina. These medullations, however, are usually irregularly-shaped and fade off into normal retina.

Persistent papillary remnants of the hyaloid artery or its sheath may have the appearance of a detachment of the internal limiting membrane. These vestiges, in contrast to the latter, extend out into the vitreous chamber and have no retinal attachment.

CONCLUSIONS

Detachment of the internal limiting membrane of the retina is frequently the sequel to lesions of the fundus, or shrinkage of the vitreous. Smaller detachments or grooves (wrinkling) are seen in edema or following atrophy of the anterior retinal layers. Detachment of the internal limiting membrane of the retina has certain characteristics by which it may be differentiated from other conditions of more or less similar appearance.

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DISCUSSION

Dr. Wilfred Fry, Philadelphia: Dr. Lyle has presented to us a subject the importance of which is not ordinarily fully appreciated, except in one phase. That phase is the relation of detachment of the internal limiting membrane and vitreous, or antecedent factors, to retinal detachment.

When we consider the anatomy of the parts of the eye under discussion, one of three possible conditions may exist. There may be, first, a membrane, properly belonging to the retina, consisting of the foot-plates of the cells of Müller and forming the only line of separation between the retina and the vitreous. Secondly, there may be a membrane properly belonging to the vitreous, forming a line of separation between the retina and the vitreous and which may be called the hyaloid of the vitreous. Finally, there may be two lines of separation, one consisting of the foot-plates of the cells of Müller, and the other of the limiting membrane of the vitreous. There has long been a discussion concerning which of these conditions exist.

The evidence that has been advanced has been confusing and has not led to any definite conclusion. As pointed out, the embryological evidence distinctly favors the existence of only an internal limiting membrane of the retina. Ida Mann makes the positive statement that "there is never anything to be seen in embryonic stages to warrant the assumption that a hyaloid membrane separating the vitreous from the retina exists apart from the internal limiting membrane, which appears to be in continuity with the two." Even as early as the 10 mm. stage, an internal limiting membrane is seen, that is more strongly united to the vitreous than to the retina, for when shrinkage occurs, the foot-plates of Müller's fibers tear away from their retinal attachments and remain with the vitreous. The conclusions of Duke-Elder in experiments upon the vitreous of animals also supports the view that the vitreous has no enveloping membrane of its own.

In the adult human eye, in the majority of preparations it is possible to demonstrate a membrane of measurable thickness, and for such preparations the view of Salzmann holds that there is only one membrane existing between the bases of the fibers of Müller and the vitreous, and that it is just as proper to term it a hyaloid of the vitreous as the internal limiting membrane of the retina.

Although the membrane appears as a continuous structure in the usual histological preparation, this continuity has been questioned. Berliner believes this continuity may be an optical illusion and states that on surface view the membrane "appears as an extremely fine network of close meshes resembling a fenestrated membrane." Preparations stained with silver carbonate indicated that the membrane was formed by fine prolongations from the branches of the cells of Müller and astrocytes.

Reference has been made to the views of Duke-Elder, who, because of ultra-microscopic and chemical studies, believed the vitreous to be a uniform gel and the border layer to be merely a surface condensation. The conclusions of Krause from a study of the chemistry of the vitreous are different. He concludes that the chemical distribution of the residual protein, which he calls vitrein, suggests that the vitreous humor is not strictly a uniform gel and that the hyaloid membrane is not a condensation of this gel but is composed of vitrein.

If the vitreous were a uniform gel, a surface condensation should be able to occur at any part of the vitreous when exposed under proper and similar conditions. Because of certain slitlamp observations made by Dr. Cowan several years ago, we made a number of preparations in which, after the lens and the greater part of the anterior segment of the eye were removed, the posterior part, containing all the vitreous, was cut in an anteroposterior meridian. This gave an anterior and a lateral exposure of the vitreous. The eyes were then put through the usual procedures and cut so that the anterior edge and the lateral cut edge of the vitreous were in the same section. These two borders had undergone the same treatment. In these preparations it was possible to demonstrate a staining and structure difference, and we believed that the existence of an anterior hyaloid of the vitreous was demonstrated. A similar procedure should be applicable to the posterior portion of the vitreous. In a further attempt in the study of the vitreous limits we have not found the use of dyes very serviceable because of the amount of disturbance invariably made at the time of injection.

When the usual clinical and ophthalmological conditions are considered, I do not believe that it is possible to differentiate between a detachment of the internal limiting membrane of the retina and a detachment of the vitreous. If the argument which the author has developed is true, and under ordinary circumstances it is, namely, that when a line of separation appears between the retina and the vitreous, the attachment of the internal limiting membrane is greater to vitreous than to the retina, then such a differentiation would be both difficult and unnecessary. Many instances of detachment of the vitreous are undoubtedly difficult to determine, due to opacities within the vitreous. On the other hand, where the vitreous is reasonably clear, it would be difficult to be assured of the position of the internal limiting membrane except in unusual instances such as have been described by Lister and others, where, in a globular detachment of the vitreous, a hole could be seen corresponding to the optic disc. Goldstein has described a case in which there was a patch of retino-choroiditis corresponding in size and position to a vitreous hole.

One of the causes of detachment of the internal limiting membrane, namely, traction, has been described and illustrated in detail. There is a

second cause which is operative in a number of cases, on which more emphasis should be placed, and that is edema. This is of importance, first in the location of the line of separation, because the edema would form on the retinal, in other words, the vascular side of the membrane, and, second, in the very early stages the edema would follow the line of the fibers of the fiber layer of the retina and thus account for the appearance of what is frequently spoken of as "tacking down of the retina" in early retino-choroidal lesions and also account for the radiating lines seen previous to the formation of a star figure in the macula.

Dr. F. Bruce Fralick, Ann Arbor: It was with much pleasure and some misgivings that I accepted the invitation to discuss Dr. Lyle's very interesting paper on "Detachment of the Internal Limiting Membrane of the Retina." I had not considered this condition as a clinical or pathological retinal entity and had not made the diagnosis either in my laboratory or clinical experience.

The outstanding example of detachment of the internal limiting membrane of the retina is seen in the easily recognized cases of subhyaloid retinal hemorrhages. The clinical and pathological picture here has long been recognized and needs no further discussion. Dr. Lyle has illustrated the pathological separation of this membrane in several retinal conditions and has suggested its possible occurrence in relation to several others. Because I had failed to recognize an antemortem separation of the internal limiting membrane of the retina in my laboratory examinations of enucleated eyes, I reëxamined serial sections of one hundred eyes in which a diagnosis of retinal disease had been made for confirmatory evidence of the condition under discussion. I found many examples of separation of the membrane from the retina as a result of fixation shrinkage or tearing of the sections during their preparation. The posterior border layer of the shrunken vitreous was often thickened by inflammatory products or showed packing together of its constituent elements to give the appearance of a separated internal limiting membrane. Many specimens illustrated the inflammatory glial or proliferative retinal bands extending from one portion of the internal surface of the retina to another, but no examples of these factors causing the internal limiting membrane to separate from the nerve fiber layer were found. Where the retina was attached to the vitreous by inflammatory retinal bands, the entire thickness of the retina was detached rather than a histological portion of it.

I obtained the impression from Dr. Lyle's discussion that the attachment was stronger between the internal limiting membrane and vitreous than the attachment of this membrane to the nerve fiber layer of the retina. I could find no examples to confirm this view and from my short series of specimens I feel that the reverse is the true state of affairs. I am under the impression that the retina will separate from the underlying pigment epithelium before it will develop a cleavage of itself as a result of an inflammatory adhesion of the retina to the vitreous. In simple shrinkage of the vitreous body, I could find no example of membrane separation — the membrane uniformly remaining with its parent structure.

I have been on a constant watch for this condition clinically since reading Dr. Lyle's manuscript and have come to the conclusion that if such does occur, as so ably demonstrated by Dr. Lyle, it must be much more easily seen pathologically than clinically. A nearly structureless, transparent membrane of one to two microns in thickness must be under very favorable conditions for observation before being seen with the ophthalmoscope, which magnifies fifteen times.

The adhesion of the internal limiting membrane to the retina at the macula must be very weak due to the absence of the supporting fibers of Müller in this region. This accounts for the large globular cystic spaces found here and explains the difficulty in obtaining histological sections of this area showing the membrane adherent to the retina proper. It is in this area that I would most often expect to find the condition described by Dr. Lyle.

Until we can demonstrate pathologically that the radiating lines about the disc and macula in retinal inflammations are due to folds or detachments of the internal limiting membrane and not due to the accepted conception of the swollen retina no longer being able to conform to the limits established by the internal surface of the sclera and choroid and the entire retina thereby being thrown into radiating folds, I feel that we must accept Dr. Lyle's views in this regard with reservations.

In spite of my inability to confirm or agree with Dr. Lyle's findings concerning detachment of the internal limiting membrane of the retina, I feel that he has presented a very interesting discussion of a condition deserving of further investigation in our clinics and laboratories.

DR. DONALD J. LYLE, closing: I wish to thank the discussers for taking part in this argument.

I should like to answer some of Dr. Fralick's questions. He speaks of the band-like fibers, proliferative fibers. Dr. Bedell refers to these fibers in his beautifully compiled and illustrated Atlas. He also calls these bundles of fibrous bands, especially in the type that he demonstrates, proliferating retinitis. I went over these pictures in his Atlas just a short time ago and I find that in at least 75 per cent of them you will find detachments of the internal limiting membrane, the membranes passing over the blood vessels.

I should like to have Dr. Fralick refer to this book to see some of these clinical cases presented by Dr. Bedell and not named by Dr. Bedell as detachments of the internal limiting membrane. I think he calls them hyaloid bands, or something of that nature.

To answer the question that the membrane, which is one to two microns in thickness, is too thin to be visible. I mentioned in my text that, due to the folding of the membrane, due, possibly, to some exudation, some cellular exudation, some other fibrous increase and thickness of the posterior vitreous, this ordinarily normal band is increased very much in thickness, which will also aid its visibility.

It is stated that not many sections are to be had of this, that in a series of 100 sections, not one was absolutely that of detachment of the internal limiting membrane. You must remember that not many of these

eyes come to section, because not many of them are enucleated. They are not the type of eye that must be enucleated. They are not taken out because of that condition.

However, in cases of hemorrhagic retinitis, and in glaucoma and other types where we have found these,—I think I have eight or ten with me—by looking over a series of 1,000 or 2,000, I have picked out possibly eight which I am absolutely sure are detachments of the internal limiting membrane. There may be another dozen which are open to suspicion.

The fact that was brought out, that the hyaloid membrane or the internal limiting membrane, whichever you want to call it, is more firmly adherent to the retina than it is to the vitreous is true if the lesion is deep. If the lesion is deep enough to involve the pigment layer, the retina is probably adherent to the pigment layer, too, and the lesion is probably thickest, tensest at the base; therefore, you cannot have detachment of the internal limiting membrane. But if the inflammation is superficial so that the tension occurs chiefly on the surface and does not occur all the way through the thickness of the retina, you have separation of the internal limiting membrane with the retention of the rest of the retina in its normal position in contact with the choroid.

If, as I showed you in one picture, there is enough inflammation throughout the whole layers of the retina, there is complete detachment of the retina. I believe those are the chief points brought up by Dr. Fralick that I wished to answer.

OPHTHALMOMYIASIS INTERNA

CASE REPORT AND REVIEW OF THE LITERATURE*

WILLIAM BANKS ANDERSON, M.D.

DURHAM, N. C.

THE TERM ophthalmomyiasis was first used in 1840 by Hope to designate the invasion of the human orbit by the parasitic larval stage of certain flies in the order of the Diptera. Of the fly larvæ that have been found in the human host, practically all have been identified as belonging to families of Muscidæ or Œstridæ, which in the larval phase, normally inhabit the tissues of certain of the Ungulates.

Salzman,[1] in 1718, recorded the first case in which these larvæ were found in the human orbit. He reported the case of a young man, who, while sleeping in the open, had both eyes literally eaten from his head. Cloquet,[2] in 1823, reported the case of a junk dealer, who, while in a drunken stupor, had both eyes destroyed by maggots. Grube and Schnee,[3] in 1853, reported two brothers, twelve and eight years of age, each of whom had both eyes destroyed by maggots, after sleeping in the open. As late of 1906, Schultz-Zehden[4] reported the case of a drunken female vagabond who had lost both eyes in a similar manner. Since Salzman's original report in 1718, more than one hundred articles, reporting more than two hundred cases of ophthalmomyiasis, have been written. The disease is described in all degrees of severity from the tragic results mentioned above to the relatively benign form which occurs so commonly in Algiers as to have acquired a local name "Thim'nis." The larvæ of this fly (Thim'nis) have no penetrating organs, and the disease is here limited to folliculosis and pseudo-membranous conjunctivitis, irritating symptoms due to the presence of the foreign body in the conjunctival cul de sac. Oronato[5] states that ninety-eight cases of this disease have been reported from Tripoli

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alone. MacCallan[6] reports thirty-two cases from Egypt. Six cases have been described in the American literature, those of Phillips,[7] Lemere,[8] Schenck,[9] Stark,[10] Bedsole and Neal,[11] and de Boe.[12]

Behr,[13] in reviewing these reports, proposed that those cases in which the larva was found external to the globe, or where the globe had been destroyed by direct attacks of many larvæ, be designated as ophthalmomyiasis externa, and that those cases in which the larva was found in the interior of the intact globe be designated as ophthalmomyiasis interna. He, furthermore, divided the latter group of cases into an anterior and a posterior group. That is to say, ophthalmomyiasis interna anterior, when the larva was found in the anterior chamber, and ophthalmomyiasis interna posterior, when the larva was found in the posterior segment. In analyzing the many cases reported to date it becomes evident that while numerous reports of the external manifestations of the disease have been made, descriptions of the internal variety are extraordinarily rare. It has been my good fortune to have under observation such a case — a man, who has been discovered to have, located in the vitreous body of the left eye, a single perfectly-formed larva, which has been identified as belonging to the order of the Diptera. This patient was seen in consultation by Dr. Wheeler. It is through his interest that I am privileged to make this report.

The patient, C. T. Mc., a white, male, insurance broker, age forty-six, was first seen on July 20, 1933, when he came in complaining of excessive pain in the left eye. His history is irrelevant, except that he is fond of sport and spends much time in the thinly-populated and marshy districts of eastern North Carolina. With the exception of a trip to Mexico (1930), three years before the onset of the present trouble, he has never been in the tropics. He dates the onset of his present illness as of July 17, 1933. On that day, during an unusually warm, sultry, and sunny afternoon, while lolling half asleep in an open motor car, being driven through the Piedmont section of North Carolina, he was suddenly roused by a light, fluttering blow in his left eye. He was holding a lighted cigar, and it was thought that ash had blown into his eye. There was only momentary discomfort, and the accident was forgotten, until recalled by subsequent events. About two hours after this incident, the patient observed that the vision in the left eye was obstructed by a dark crescentic-shaped object about four millimeters long

and one-half a millimeter wide, which seemed to be suspended in space an inch beyond the eye. He was unable to dislodge this object by pushing back his eyebrow, moving his hand in front of the eye, or rubbing the eye. After remaining in his line of vision for forty-five minutes, this object suddenly disappeared. On the following afternoon, the eighteenth, while taking a bath, the left eye began to sting. The sensation lasted for a very short time. In this instance, the discomfort was attributed to having got soap in the eye. A few hours later, he began to experience severe and continuous pain in this eye. On the following morning, the nineteenth, he consulted an oculist, and was told that he had a conjunctivitis. Reassured and relieved of the pain, he returned to his office and performed his routine duties. During the night of the nineteenth, he was awakened by a pain in the left eye so severe that morphine gave only temporary relief. The patient was first examined in the Duke University Clinic on the morning of the twentieth. At that time he appeared to be in no great pain, although very obviously showing the effects of the previous night's experience. At this time, the eye felt uncomfortable and there was a slight stinging sensation which he could not localize. Recognizing that the case presented many unusual features, he was given an exhaustive examination with both ophthalmoscope and slitlamp. Findings at this time were entirely negative. Except for a moderate diffuse conjunctival injection, there was nothing to be found that could be remotely associated with the paroxysms of pain of which he complained. An attempt was made to reassure him, and he was asked to report the following day. However, that night he again began to have pain in this eye. Again the pain was so severe that morphine gave only temporary relief. He was admitted to the hospital the following morning. When first seen he appeared to be in shock. He was nauseated and had been vomiting. He was holding the left side of his face in both hands and appeared in the most abject misery. He complained bitterly "that hundreds of fine red-hot needles were being jabbed into his left eyeball." A searching examination of his eye, undertaken at this time, revealed a generalized moderate injection of the conjunctiva. The pupil was slightly and irregularly dilated. The slitlamp revealed a somewhat fuzzy and swollen iris with a slightly opaque aqueous ray. Ophthalmoscopic examination showed a slightly turbid vitreous. There was a slight discoloration in the retina in the lower nasal quad-

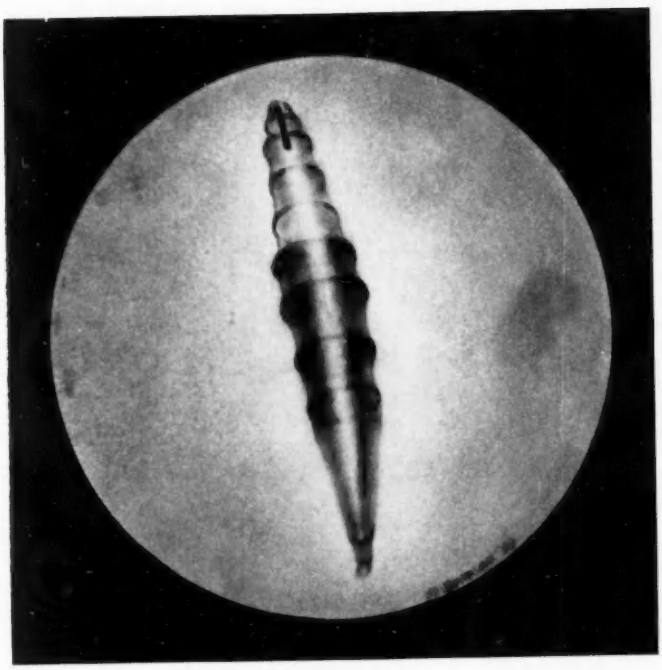

FIG. 1 — (Anderson)

(OPPOSITE P. 220)

rant, and in the lower temporal quadrant there was a small deep choroidal hemorrhage. The tension was normal. Field studies showed no defect, no scotomata. There was no unusual pain on pressure or motion. His vision was blurred and there was considerable, though not excessive lacrimation and photophobia. The most striking feature of the picture was pain, excessive, out of all proportion to the appearance of the eye. A tentative diagnosis of iritis was made, and the usual treatments for this condition were instituted.

The acute pain gradually subsided during the following twenty-four hours, but did not entirely disappear. The nausea and stabbing sensation persisted until the night of the twenty-third, when, while the nurse was applying hot compresses to the eye, the sensation suddenly and completely disappeared. The eye has remained without pain from that time. Immediately, when the pain disappeared, the patient again became conscious of an object, crescentic in shape, which obscured his vision. This object had the same general appearance as that noted immediately after the blow on the seventeenth, except that the curve was not now so acute as when first seen.

On the morning of the twenty-fourth, one immediately and without the slightest difficulty observed the astonishing picture shown in figure 1 (colorplate). Located in the more anterior portion of the vitreous body, and lying in the vertical plane, was a beautifully-symmetrical, amber-colored, tapered and segmented, cigar-shaped foreign body, in length about seven millimeters, and about one-tenth as broad. Extending longitudinally through the middle was a lighter streak, bright golden in color. The parasite was divided horizontally into nine evenly-spaced and one elongated and one short terminal segments. The segmenting lines showed up a much darker color, in which were interspersed, at regular intervals, more highly refractile dots, giving these rings the appearance that filigree work had been imposed upon them. Entomologists state, these rings are really rows of spikes or cilia. At the upper blunt end, the superior border of the first segment is surmounted by a ring of fine cilia. This is best shown in the more schematic free-hand sketch, made at the time of discovery (Fig. 2A). Inserted in this end, and extending down through the third segment, is a Y-shaped stigma, each distal end club-shaped. The details of the inferior end have never been clearly seen. Figure 2B is a photograph of a definitely determined Gasterophilus hæmorrhoidalis, which is inserted here to

show the similarity between the Gasterophilus and the larva. The general physical and laboratory examinations were entirely negative. There were no parasites in the stools, there was no eosinophilia, no general glandular or splenic enlargement — in short, nothing that pointed to a general invasion of the host.

FIG. 2A—(Anderson)

FIG. 2B—(Anderson)

Figure 2B — Tentatively determined as first stage nose bot, Gasterophilus hæmorrhoidalis, removed from arm of man at or near Fargo, North Dakota.

No one could possibly have mistaken the fact that this foreign body was a larva. An effort was immediately made to learn something of the identity and, possibly, the treatment of this condition. Dr. A. S. Pearse, of the Department of Zoology, Duke University, gave us our first real clue as to the condition. He stated that infestation of the sinuses of cattle with the larva of the Œsterus ovis was not uncommon, and that in his opinion, one of these larvæ had probably found its way into the patient's eye.

Repeated examinations of the patient always showed the larva in the same position. No motion was ever seen either by the examiner or the patient, who seemed to have as good as or better view of the organism than the examiner. There was no further pain. The vision, which had been blurred, cleared very rapidly. Except for the annoying presence of the "zeppelin," which occasionally came into his line of vision, there was no further complaint from the patient. Naturally, being very much

FIG. 3.—(Anderson)

concerned as to the ultimate result, and not caring to take the entire responsibility of advising him on so important a matter, a consultation was suggested. The patient was sent to Dr. John M. Wheeler on July 30. Dr. Wheeler advised against surgical intervention. The subsequent course of the disease has proven the soundness of his advice. No change was noted in the appearance of the larva until the last of September, 1933, when it was noted that the shape was gradually changing, the upper part of the animal was becoming more swollen, and the more dependent half was shriveling. This process has continued, and now the parasite is shrunken and crumped into a more angulated

mass. The patient states it looks as if its back had been broken. It is thought that there is no question but that the larva died immediately after gaining entry into the globe and that now the resistant chitinous skeleton is being absorbed. Tonometric and field studies have remained negative. His vision remains 20/20 in each eye. Figure 3 is a drawing made in August, 1934, showing the change that has taken place in the organism in the past year.

It is interesting to note that the patient has never experienced the least discomfort since the larva became visible to him. It seems fair to assume, then, that the pain was due to the passage of the larva through the scleral coat, and that once past, all energy being consumed, it met death in the vitreous body. This is the only case on record in which the larva was allowed to remain in the eye in which the globe was not destroyed. Since Baer's work on maggots in the treatment of osteomyelitis, we have learned something of the proteolytic ferments generated by these larva, and it is impossible to believe that a living larva secreting this ferment could have remained in the eye without destroying its highly sensitive structure.

The identity of the larva remains in doubt. Entomologists have labored under the handicap of having to make the identification from drawings alone.

F. C. Bishop, Chief Entomologist of the United States Department of Agriculture, writes, "It is impossible to make a definite identification from the drawings, but I would say that the drawings resemble the first instar of the common horse bot, Gasterophilus intestinalis, more closely than any of the other forms which is commonly concerned in the causation of myiasis in man. The history of the case tends to exclude this species, infestation by which ordinarily starts with the deposition of eggs, and these require several days to hatch. Ocular myiasis in man is usually caused by the sheep bot fly, Œstrus ovis, but the drawings which you submit do not closely resemble this species. * * * I should like to give a little further consideration to the possibility of the trouble being due to the larva of some sarcophagid fly. Most of the Sarcophagidæ are larviparous and this would explain the sudden pain and apparently rapid progress of the larva, which could not be explained if the larva had started as an egg."

Dr. F. W. O'Connor, who saw the drawings at the request of Dr. Wheeler, states, "Owing to its geographical distribution,

the appearance of the drawings and, especially, the pencilled one, I am inclined to believe that the maggot you are dealing with is that of the fly Cochliomyia (Chrysomyia) macellaria, in common parlance, the screwworm."

C. H. Currin, Assistant Curator of the American Museum of Natural History, to whom drawings of the larva were also submitted, expressed the opinion that "the larva was almost certainly a species of Gasterophilus and most probably Gasterophilus intestinalis." He states that "the larva of this species hatches immediately upon coming in contact with moisture and would lose no time in working its way toward the back of the eyeball, later moving into the eye." Currin also makes the significant statement that "it is very likely that much of the literature is erroneous in regard to the identity of the species of flies concerned, since we know so little about the larvæ, and it is only recently that it has been possible to definitely identify Œstroid larvæ in the first stages."

None of these opinions as to identity are compatible with the known facts of the history. That is to say, the fly must have been viviparous, since the patient saw almost immediately after the blow a crescentic-shaped object, which, as described by him, must have been at least three or four millimeters in length, possibly longer. This would agree with the identification of O'Connor, but the Chrysomyia is not larviparous. The Œstrus ovis is larviparous, but, apparently, has no penetrating organs which would enable it to penetrate the sclera.

Eighteen other cases of ophthalmomyiasis interna have been reported. In ten of these cases, the larva was found in the anterior chamber. The other eight cases were found in the posterior segment, either in the vitreous or in subretinal exudate. Tables 1 and 2 represent a summary of these cases.

In 1900, Krautner[14] (Klagenfurt, Austria) reported the first case of ophthalmomyiasis interna. He found in the anterior chamber of a nine-year-old Austrian girl a motionless, semitransparent, greyish-white worm-like figure, one and one-half millimeters broad and about eleven millimeters long, divided into seven cross segments. A few months previously, during the summer, she had been struck in the eye by an insect (wasp or fly). At the time of the first examination, the reaction of the eye was very slight. The conjunctiva was reddened, the aqueous cloudy, there was some ciliary injection. By January, she had

TABLE 1 — *Ophthalmomyiasis Interna Anterior*

No.	Year	Author	Geographical Distribution	Age	History and Physical Findings	Results	Identity of Larva
1.	1900	Krautner	Klagenfurt, Austria	9	First seen in Jan. — Periodic inflammation since insect bite over lid in summer. Tension plus. Hypyon. Cornea and aqueous cloudy. Moderate ciliary injection. Larva in anterior chamber.	Removal attempted. Death from chloroform.	Hypoderma bovis.
2.	1901	Stahlberg	Sweden	5	Duration five months. Pain and irritation.	Extracted. Recovery.	Hypoderma bovis.
3.	1904	Ewetsky and Von Kennel	Dorpat, Russia	5	Seen in Feb. Onset Sept. Duration 5 months. Exacerbations of inflammation. Pain. Cloudy iris and cornea. Turbid aqueous. Larva in anterior chamber.	Extracted. Vision 3/10.	Diptera.
4.	1908	Thomas and Parsons	England	3	Onset: Indefinite, several months. Photophobia, irritation, discolored iris and pupil, turbid iris. Larva in anterior chamber.	Enucleated.	Larva of Blow Fly or Sarcophaga.
5.	1922	Magiorre	Italy	16		Extracted.	No note.
6.	1923	Bietti	Italy	10	Onset: October. Photophobia, pain. Descemetitis, iritis. Larva in anterior chamber.	Aspirated.	Hypoderma. Probably H. Lineatum.
7.	1924	Blessig and Kuriks	Dorpat, Russia	3	Recurrent attacks of phlyctenular kerato-conjunctivitis over a period of months.	Extracted. Complete Recovery.	Hypoderma bovis or Rhinoestrus bovis.
8.	1927	Hartman	Emden, Germany	5	Onset: October. Irritation for 3 months. High grade irido-cyclitis. Larva in anterior chamber.	Extracted. Vision 1/5.	Probably Hypoderma bovis.
9.	1929	Barczinski	Allenstein, Germany	6	Seen in February. Duration 10 days. Pain and loss of vision. Hypotonia, deep anterior chamber. Larva in anterior chamber.	Extracted. Vision 2/50.	Hypoderma bovis.
10.	1930	Grungarten	Russia		Article not available.		

TABLE 2 — *Ophthalmomyiasis Interna Posterior*

No.	Year	Author	Geographical Distribution	Age	History and Physical Findings	Results	Identity of Larva
11.	1913	Hess	Munich, Germany	4	Examined December. Onset insidious, 1 month. Painful at intervals. Slight ciliary injection, cloudy cornea — detached retina. Diagnosis: Tuberculosis.	Enucleated.	Hypoderma bovis.
12.	1917	von Schmidt zu Wellenberg	Klagenfurt, Austria	5	Examined February. Onset 1 month previously. Cloudy cornea, suppurative choroiditis, detached retina. Tension 35. Traumatic glaucoma, probably due to percussion cap?	Enucleated.	Diptera.
13.	1920	Behr	Kiel, Germany	7	Onset: August — 4 months duration. Tension 38. Phlyctenules — cloudy cornea. Subretinal larva. Tuberculosis.	Enucleated.	Diptera. Probably Hypoderma bovis.
14.	1925	Purtscher	Klagenfurt, Austria	9	Onset: July. Pain, loss of vision, deep anterior chamber. Iris discolored.	Larva Extracted. Vision lost.	Hypoderma bovis.
15.	1926	Zeeman	Amsterdam, Netherlands	6	Duration 4 months. Retinal separation. Subretinal larva.	Enucleation.	Diptera.
16.	1931	Archangelsky and Braunstein	Moscow, Russia	4½	Pain beginning 1930. Seen 2 to 3 weeks after onset. Observation 2 months. Exacerbations. Hypopyon, hypotonia, deep anterior chamber. Detached retina. Diagnosis: Tuberculosis.	Enucleation.	Œstridæ.
17.	1933	DeBoe	Miami, Fla., United States	48	Onset: May. Rapid loss of vision. Larva presents through optic foramen and moves into vitreous.	Optic Atrophy.	Chrysomia.
18.	1934	Ennema	Amsterdam, Netherlands	3	Onset: December. In May increased tension. Tumor protruding into vitreous. Diagnosis: Glioma or pseudo-glioma. Subretinal larva.	Enucleation.	Diptera.
19.	1934	Anderson	Durham, N. C. United States	46	Onset: July, with excruciating pain. Physical examination negative. After few days larva found in vitreous. No reaction in tissues of globe.	No damage.	Gasterophilus equi.

developed increased tension, hypopyon and iridocyclitis. At this time an attempt was made to remove the larva; unfortunately, the patient died under chloroform narcosis. The larva was definitely identified as the Hypoderma bovis, one of the Œstrids.

In 1901, Stålberg[15] (Jönköping, Sweden) reported a case of a five-year-old girl who had had an irritated eye for five months (September to February). Symptoms had gradually become more pronounced and recently the exacerbations of pain had been associated with attacks of vomiting. Examination revealed a segmented, greyish, transparent object lying in the lower part of the anterior chamber. This figure was about one centimeter long and was tapered at both ends. The larva was extracted and identified as the first stage of Hypoderma bovis. The patient made a complete recovery.

Ewetzky and Von Kennel[16] (Dorpat, Russia), in 1903, reported the case of a five-year-old boy, who, since September, had been having symptoms of irritation in one of his eyes. He was seen five months after the onset of symptoms, characterized by exacerbations of pain, photophobia, redness. Examination revealed a swollen iris, a cloudy cornea, turbid aqueous, and, lying at the bottom of the anterior chamber, a parasite about six or seven millimeters long and one and one-half millimeters broad. This was cylindrical in shape and had tapered ends. The parasite was removed. Von Kennel was unable to identify it definitely, but was convinced that it was a larva of the order of Diptera. He suggested that possibly, due to the unusual environment, the larva had undergone mutations which made further identification impossible. Three weeks after removal, the patient had recovered 3/10 vision.

In 1908, Thomas and Parsons[17] (British Isles) reported the case of a three-year-old boy in the anterior chamber of whose eye was found a greyish-yellow worm eleven millimeters long and two millimeters broad. The patient's mother had noticed that the child had developed the laughable habit of starting suddenly as if he had suddenly seen something strange, then, thrusting the hand before the left eye as if to brush something from his line of vision. About three and one-half weeks before the patient was seen by Thomas, the mother had noticed that "the middle of the left eye looked somehow different." The child had, also, about this time, begun to develop irritable spells, occasionally holding the eye as if in pain. Thomas, examining the

eye under an anæsthetic, found the cornea to be perfectly clear, with very slight ciliary injection. The anterior chamber was deeper than normal, the aqueous faintly turbid. The iris was altered from a blue to a dirty grey color; there was hypotonia, the pattern faintly fogged. The pupil was irregular and bound to the lens. In the lower outer quadrant of the anterior chamber, Thomas saw a cylindrical worm twelve to thirteen millimeters in length, tapered at both ends and broadening to a width of two millimeters in the center. The body was of a greyish-yellow color, and was divided into eight clearly-defined segments. The eye was enucleated. Shipley, at Cambridge, thought that the larva was that of the blow fly, but might be that of one of the Sarcophaga.

In 1917, Magiorre,[18] in Italy, reported finding in a sixteen-year-old boy the larva of one of the order of the Diptera, lying with one free end extended vertically into the anterior chamber, while the other end was adherent to a small leukoma near the limbus. This larva was removed by gentle and continuous traction on the attached end. No further information was available as to identity of the larva or the fate of the patient.

In 1923, Bietti[19] (Italy) reported the case of a ten-year-old girl, who, in the latter part of October, while cutting a piece of wood, had this slip from her hand strike her in the right eye. The eye had shown signs of progressive irritation since that time. She was seen in the clinic the last of November. At that time, vision was limited to finger movements in the injured eye. There was markedly increased tension, the ciliary region was injected, the cornea steamy. There was a posterior descemetitis. The aqueous was turbid. The pupil was narrowed, but reacted weakly to light. Lying free in the aqueous in a vertical plane was a cylindrical body, which moved passively when the eye was moved, although keeping at all times the vertical plane. The object was about eight millimeters long and one millimeter broad. It was greyish-white in color and clearly divided transversely into eight segments. A linear incision was made and the body aspirated from the anterior chamber with a pipette. The larva was identified as the Hypoderma genus, probably lineatus.

In 1924, Blessig and Kuriks[20] (Dorpat, Russia) removed a living larva from the anterior chamber of a three-year-old girl. This child had been under observation for several weeks suffering with what was thought to be recurrent attacks of phlycten-

ular conjunctivitis. A few days after the larva was discovered, it lost its motility and came to rest in the lower angle of the anterior chamber. An incision was then made and the larva removed. After a secondary rise in tension, associated with hemorrhage into the anterior chamber, the eye made a complete recovery. The larva was three and one-half millimeters long and one and one-half millimeters broad. It was divided transversely into nine segments. This larva was identified as either the larva of Hypoderma bovis or that of the Rhinœstrus nasalis.

Hartman[21] (Emden, Germany) reports the case of a boy, aged five, whom he first saw in December of 1926. This boy had had symptoms of irritation in the offending eye for a period of three months. A diagnosis of iridocyclitis was made. He was kept under observation until the third of January, when the presence of a parasite began to be suspected. By this time, a foreign body could be seen in the pupil, the lens being partially dislocated. By the tenth of January, the larva had made its way into the anterior chamber. An incision was made at the limbus, and in the rush of fluid the head of the larva presented in the incision. It was grasped with an iris forceps and removed. It was classified as probably Hypoderma bovis. The child's eye immediately became white and quiet. Vision after removal was noted as 1/5.

Barczinski[22] (Allenstein, Germany) reported the case of a six-year-old boy first seen in February. There was a history of a red and inflamed eye with rapid loss of vision during the past ten days. There was no history of prior injury or discomfort in the involved eye. Examination at this time revealed a high grade hypotonia, ciliary injection, deepened anterior chamber, and cloudy vitreous. In the anterior chamber was a foreign body about one centimeter long and two millimeters broad. It was removed and identified as the larva of a gadfly. In this case, the parasite was dead on removal. The vision in the invaded eye was reduced to 2/50.

Grugarten's case report[23] was not available, and is mentioned here merely for the sake of completeness.

In 1913, Hess[24] (Munich, Germany) reported the first case of the interna posterior variety of this disease. This case also comes from Germany. This patient was a four-year-old girl first examined by Bergmann towards the end of December, about two months after the onset of the symptoms. The eye, after a

period in which there had been some redness and pain, showed at the time of this examination slight ciliary injection and cloudiness of the cornea. A month after the first examination it was noticed that the pupil had acquired a greenish reflex and a descemititis. A diagnosis of tuberculosis or glioma was made. Enucleation was then performed. Pathological report revealed a detachment of the retina and under this a larva one and one-half centimeters long. This was identified by Gruenberg as the larva of the genus Hypoderma of the first stage. In this case, the modifications in the anterior segments of the eye were very slight. There was purulent inflammation in the retina and choroid. From a study of the pathological specimen, Hess believed the larva found entrance into the globe by way of the bloodstream.

The case reported by von Schmidt zu Wellenberg[25] (Klagenfurt, Austria) concerned a five-year-old child. In this instance, the larva was found at pathological examination after removal of the eye. The enucleation was performed on account of a suppurative chorio-retinitis, presumably from a percussion cap. The child had, on examination, hypertonia, cloudiness of the cornea, and amaurosis, associated with separation of the retina. Subluxation of lens and symptoms had persisted for five weeks (since February). The larva was not identified other than to state that it belonged to the order of the Diptera.

Behr[26] (Kiel, Germany), in 1920, reported the case of a seven-year-old boy who had first begun to have trouble with his eye in August, 1918. A swelling in the left cheek was first noticed. This lasted for three days. Following this there was a swelling noticed in the palpebral conjunctiva. Twelve days after the onset there was noted, at the corneal margin, "a phlyctenular irritative condition," with normal findings in the interior of the eye. This condition lasted for three weeks, after which there was noticed at a certain point a gibbous prominence in the sclera. Subsequent to this, mobile bodies (vitreous opacities?) were seen in the vitreous, and hemorrhages around the papilla. A little later, separation of the retina occurred. Following this, symptoms of irritation disappeared until the first of December, when increased tension and intense inflammatory symptoms necessitated enucleation. Postoperative impression was glioma or tuberculosis of the choroid. Microscopic examination revealed a severe choroiditis, which had caused separation of the retina.

Under this was found a Diptera larva, which was identified, at least with great probability, as the larva of a Hypoderma bovis.

Purtscher[27] (Klagenfurt, Austria), in 1925, reported a case unusual in that the diagnosis of ophthalmomyiasis interna posterior was made preoperatively. Possibly he was somewhat more alert to this possibility because two of the previous cases reported came from his clinic, those of Krautner and Schmidt-Wellenberg. His case, a nine-year-old boy, who stated that on the night of July 31 he became conscious of severe burning in the left eye, and noticed the following day that vision in this eye was very much impaired, although the pain had entirely disappeared. The condition had not changed before the first examination on August 8. On examination, the involved eye was found to be free from injection, the cornea clear, the anterior chamber deeper than normal and the iris showing a greenish discoloration. The pupil was regular and showed reaction to light. Vision was limited to hand movements eccentrically. There was a massive separation of the retina. Beneath the detached retina a clearly defined foreign body was easily seen, although the finer differential points could not be made out. It was assumed that this was a case of subretinal cysticercus, although there was nothing in the history to indicate such a development. While keeping the boy under observation, there was discovered, on August 25, a shiny yellowish-white larva lying upon the detached retina with the head and four segments clearly visible. Along its middle was a brilliant golden stripe. During an hour's observation, the constant suctorial movements of the animal and contraction in a longitudinal direction were clearly visible. On the twenty-eighth, this larva was removed through a scleral incision. Immediately after the knife was withdrawn there was an escape of fluid and the head of the larva presented in the opening. It then crawled of its own volition out onto the conjunctiva. The irritated condition of the eye had largely cleared by the time of the discharge of the patient on the fifteenth of September, although vision remained limited to hand movements. One and a half years later, the eye remained the same, except that a cataract had developed. Purtscher states that the bulb at no time showed any signs of inflammation other than that induced by operative interference. Examination of the larva showed it to belong to the Œstridæ, undoubtedly Hypoderma bovis, in the first stage of development.

Zeeman[28] (Amsterdam, Netherlands), in 1926, reported the case of a boy of six years with progressive blindness in the left eye accompanied by comparatively few other symptoms. After four months' observation, on account of pain, enucleation was done. Examination of the globe showed subretinal larva.

Archangelsky and Braunstein[29] (Moscow, Russia) reported the finding of a dismembered larva of the Œstrid family in the enucleated eye of a four-and-a-half-year-old girl. This patient first came under observation in the beginning of 1930, suffering from pain and irritation of the eye of three weeks' standing. At examination, the entire eye showed high grade inflammation, hypopyon, hypotonia, and iridocyclitis. The fundus could not be seen. A diagnosis of chronic iridocyclitis of unknown etiology, possibly glioma or tuberculosis, was made and the patient asked to report for observation. After two months, the eye was removed, with the finding reported above.

Dr. de Boe,[30] of Florida, in 1933 made a preliminary report on the only case of this type that has been known to occur on the North American continent. His patient was a man of forty-eight years, who came into the office because of a sudden attack of blindness in the right eye one hour before. His past history was negative except that a few years previously he had lived in the tropics. He had never had any tropical disease nor any other illnesses. He had not been struck in the eye by a fly, nor was there any other injury to the eye. There had been no break in the skin except for a tick bite a few weeks previously. Mateson states that certain larva of the Diptera (Dermatobia hominis) are normally transferred by ticks, so this fact may be significant. Dr. de Boe had the good fortune to watch the larva break through the nerve head and move into the vitreous body. In this case, at least, there can be no question as to the immediate portal of entry. At the time of Dr. de Boe's preliminary report the patient was preserving the eye, but there was complete loss of vision.

In 1934, Ennema[31] (Amsterdam, Netherlands) reported the finding of a subretinal larva in the enucleated eye of a three-year-old boy. At Christmas, 1931, the mother had first noticed that the child had "a dull expression." On examination in May, 1932, examination revealed: Normal cornea without keratitic precipitates or circumcorneal injection, shallow anterior chamber, eccentric and fixed pupil, atrophy of iris, posterior synechiæ, yel-

lowish-green fundus reflex, with tumor protruding into the vitreous body, increased tension. Preoperative diagnosis was pseudo-glioma or glioma. The past history was non-contributory.

COMMENT

In reviewing these cases, one is impressed with the fact that the positive identification of the offending larva is difficult, often impossible. In six of the eighteen cases, a definite classification of the Hypoderma bovis of the Œstrid family is made. In three of the remaining cases, the larva is classified as probably Hypoderma bovis. There are, thus, nine cases more or less identified as having as the causative agent the Hypoderma bovis. In four of these cases, the history and appearance of the eye does not indicate the route by which the larva entered the eye. There is, on the other hand, very definite evidence that in the remaining four cases the entry was direct from the conjunctival sac through the sclera. If this identification of the larva is correct, we must assume that the egg was laid by the fly in flight, and that the egg remained in the conjunctival sac sufficiently long to hatch. This would require several days, and it is difficult to visualize such a foreign body remaining in this position for this length of time. In the normal life cycle of this species, the egg is laid on the skin of the cow, it is then licked off by the host, and carried to the esophagus and stomach, where it is hatched into the first stage. This first stage larva begins to migrate always in an upward direction. It, thus, in the normal host, after some months, comes to lie under the skin about the head, where it undergoes further development. It is claimed by some that the larva must find its way into the human eye in somewhat similar fashion. In some of the cases reported above, the time element might allow for this long journey. But in the majority of the cases reported there is too short an interval between the onset of the disease and the discovery of the larva. If the egg had followed its normal development in the host, we could not expect to find the larva for many months after its ingestion. Dr. de Boe's case is the only one in which the larva is definitely known to have come into the eye either through the posterior foramen by way of the nerve sheaths or by way of the bloodstream, although Hess surmised that the larva in his case gained entrance into the globe in a similar manner. Dr. de Boe's case is also the first case of ophthalmomyiasis interna to occur in an adult.

The cases of de Boe and Hess excepted, the balance of evidence, as emphasized in Magiorre's case, points to the entry of the larva into the globe by direct penetration of the sclera, for which act they have highly efficient organs. Brauer placed a larva of the Hypoderma on his arm and found that it had burrowed into the skin before he could adjust a magnifying glass. In this connection, the Germans have stressed the fact that all cases have occurred in children, Magiorre's case excepted, under ten years of age. They stress the fact that in children the sclera is less able to withstand the attack of the larva, inferring that the adult's sclera does withstand such assaults. This argument is refuted by the case reported in this paper. If the larva is carried by the bloodstream, or migrates through the blood vessels, it would seem that the vascular bed of the child, being smaller, would offer more resistance than those of the adult. If the entry is directly through the sclera, the fact that only one larva is found in all the reported cases remains unexplained, as it seems improbable that only one larva was deposited. It is, also, rather strange that these larvæ, having penetrated the highly resistant sclera, should pause beneath the retinal layer rather than seek to penetrate further. While one must admit the possibility that the larva in some of these cases was hatched in the human host, in most cases the evidence seems to point to the larva of a viviparous fly, which, being deposited on the host, immediately begins to bore into the tissue. Von Kennel and others have pointed out that the identification may be made more difficult by the modifications which take place in the development of the larva as it moves into a strange environment, and is forced to adapt itself to the nourishment available in this area.

It would seem that, in reality, the symptoms, signs, and prognosis of ophthalmomyiasis depend more upon the viability of the larva, and the position which it finally selects in the eye than upon its identity.

In the anterior chamber, these larvæ apparently can exist for months with little damage. Behr feels this is due to the rapid change of fluid in the anterior chamber, which carries away the excrement. He feels that the parasite here finds in the aqueous an inadequate nourishment which modifies and perhaps prevents further development. On the other hand, in the serum of the subretinal fluid, high in albuminous content, the larva

finds an ideal environment. In this area the fluid exchange is slow and toxic products accumulate, ultimately causing the loss of the eye.

Although the German writers claim that the ophthalmomyiasis interna presents points which characterize it as a definite clinical entity, one has difficulty in finding any such conformation of symptoms and signs. In ophthalmomyiasis interna anterior the diagnosis is, of course, easy when the larva has appeared in the anterior chamber, where it is visible to the naked eye. On the other hand, the symptoms preceding this appearance are quite misleading. In two of the cases reported above, a tentative diagnosis of phlyctenular conjunctivitis was made.

In ophthalmomyiasis interna posterior the symptoms and signs are even more confusing. Purtscher and de Boe report the only two cases, prior to this paper, in which the larva has been seen alive in the posterior segment. In the other cases of ophthalmomyiasis interna posterior the diagnosis has been made from the enucleated eye, the preoperative diagnosis favoring tuberculosis or glioma.

The prognosis in this disease would seem to depend upon the position of the larva. In those cases in which the larva is found in the anterior chamber, surgical intervention, as may be seen from a perusal of the tables, may be attempted with a fair chance of success. In the posterior variety, Purtscher alone has been able to extract the larva and save the globe.

It is interesting to note that while these cases are of great rarity, three of them have been observed at Klagenfurt, two at Dorpat and two at Amsterdam.

SUMMARY

A foreign body identified as a fly larva is reported in the vitreous body of a forty-six-year-old patient. This case, with that of de Boe, who has reported the only other case known to occur in North America, differs from all cases reported in European literature in that both of the American cases were adults, while all of the European cases were children. The European writers have previously laid stress upon the fact that the sclera of the adult was too resistent for the penetrating organs of the offending larva. The present case report also differs from all previous cases reported in that there has been no damage to the ocular apparatus, and that surgical intervention has been unnecessary.

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DISCUSSION

DR. M. P. DE BOE, Miami: Doctor Anderson has indeed presented a thorough digest of this subject in his excellent paper. In addition to the scientific data which he has given us, and which is already history, he has added a new page to the history of ophthalmic medicine.

In my case, in which I saw a Diptera larva crawl out of the disc into the vitreous in May, 1933, which case was reported in the Archives, there is very little to be reported now. It is interesting for that fact. The larva was in the same position two weeks ago, when I saw the patient last, as it was when it stopped moving seventeen months ago. It is in a perfect state of preservation in its living sepulchre. Every segment can still be seen clearly and the sucking organ is probably more prominent.

The eye is in perfect condition with the exception of a complete optic nerve atrophy. The conjunctiva is normal; cornea clear. Iris normal and pupil has perfect consensual reaction. Media are all clear and the tension is normal.

With all the available data before us it is still very difficult to construct a syndrome of this disease entity. There are, however, a few points of general interest to be observed. With the exception of the case of Kampherstein, there is not a case which showed bacterial infection, and his case became secondarily infected by a fungus. This supports the observation of the late William S. Baer, that maggots have on their bodies an antiseptic substance.

You will note that there is not a case of sympathetic ophthalmia reported in these cases. However, most of the eyes were enucleated early.

The blood pictures are not given in any of these cases, except in Doctor Anderson's and mine, which were essentially negative. M. C. Ennema of Amsterdam reports a case in the August issue of Archives, in which there was an eosinophilia.

Another interesting point is that in every case reported, except Doctor Anderson's and mine, the eyeball was destroyed. In all these cases the larvæ remained attached to some solid tissue, where they could get nourishment and continue their destructive work. In each of our cases, the

larva became disengaged from solid tissue and floated free in the vitreous, where it could not get sufficient nourishment and oxygen to live, and became an aseptic foreign body.

An acutely inflamed eye with negative bacterial findings and negative blood picture, which is not due to any well recognized disease, is certainly suggestive of ophthalmomyiasis.

Dr. Martha Lyon, South Bend, Ind.: I simply wish to present a case that appeared in the office September 12, 1934. This man, about thirty-five, working in a tire shop, was struck in the eye at ten a.m. by a fly. At four p.m. he was in the office to have something removed from his eye, thinking he had something in it.

My husband, a parasitologist, Dr. Marcus Lyon, saw the eye and, with a 40-diopter lens and good illumination, removed from the conjunctical sac ten larvæ and saw one go down in the tear duct. These were immediately identified under the microscope as Œstrus ovis, the larvæ of the sheep gadfly. They measured 1¼ mm. long and 4/10 mm. wide.

The sheep gadfly selects an eye, especially of the sheep, in which to deposit its larvæ, deposits them alive, not as eggs, and the nasal cavity is supposed to be the cavity in which the flies further develop.

I report this case, because I saw this larva under the microscope last night. It is quite different from the one on the screen. This is common in Russia, in Siberia, somewhat more rare in Italy, France and Spain.

OIL CYST OF THE ORBIT WITH CARCINOMATOSIS

ARTHUR C. JONES, M.D.
(By Invitation)

BOISE

OIL CYSTS of the orbit are not uncommon. They belong to the group of dermoid cystic tumors. There are very few cases of orbital tumors where a positive diagnosis of the true nature of the tumor can be determined before explorative incision has been made; this is especially true if the growth be deep-seated.

Henry G. Connell[1] mentions that "Berlin had collected seventy-three cases of orbital tumors, twenty-three of which were undoubtedly dermoid cysts."

In a rather careful perusal of the literature, I could find no definite reference to an orbital oil cyst of dermoid origin having become malignant. However, Berlin[2] does mention that a degenerative dermoid cyst may be the point of origin of an epithelioma. The literature contains many reports of malignant tumors of the orbit. Benedict,[3] in his comprehensive article, entitled, *Tumors and Cysts Near Apex of Orbit*, reported eight cases, all of which were malignant, but not of dermoid origin.

Most histories of oil cysts have their beginning in childhood and the cysts gradually increase in size until it becomes necessary to remove them in order to save vision. Henry G. Connell[4] gives a long description of a compound oil cyst of the orbit which began in childhood, and the eye had to be enucleated in order to remove the entire cyst. V. Camp[5] reported cases of two dermoid tumors in adults which had been noticed in early childhood and gradually increased until they had to be removed. In de Schweinitz'[6] *Diseases of the Eye*, "Berlin divided orbital cysts into two principal groups, cephaloceles and true cysts. True cysts may be classified as cysts from constriction, which are derived from congenital meningoceles (Klingelhoffer) as extravasation-cysts — that is to say, blood-cysts, hematomas, etc. — as exudation-cysts, which are very rare; as dermoids, which are

the most frequent cystic tumors growing in the orbit, as mucous-cysts, which may communicate with the nose, and as echinococcus-cysts."

In Rasquin's[7] article, entitled, "*Multiocular Internal Dermoid Cysts of Orbit,* (one oily), he begins by saying that the following observations appear interesting enough to warrant their publication.

"A child, six years old, was brought to my consultation room by the mother, who stated that for several months a tumor-like projection had made its appearance in the internal angle of the right orbit, and that the mass was growing so rapidly that she had become alarmed. My examination shows a tumor occupying the internal third of the right upper eyelid, which descends full-length from the base of the nose parallel to the lacrymal sac. The tumor is about the size of a small walnut, but it is not adherent to the skin. It is not freely movable and seems to bury itself into the orbit. A puncture made with a large needle on a syringe does not draw forth any liquid, but the needle has an oily glow and is covered with degenerated greasy mass after its removal.

"The eye is slightly exophthalmic and is displaced down and out; vision is normal; the fundus is normal." He states, "We accepted the diagnosis of a dermoid cyst of the orbit and decided to excise the tumor. A curvilinear incision is made beneath the orbit running up to the internal angle of the orbit and an encapsulated tumor mass is uncovered which is easy to dissect. The tumor is found to be divided into partitions; the anterior one passes in front of the pulley of the superior oblique and extends downward. The other extends up towards the back of the orbit; both of which are removed." He further states that "upon inspecting the wound a second tumor of an entirely different appearance is found. It appears as a whitish-gray mass, spherical in shape, and is removed in capsule. Both tumors lie outside the muscle funnel.

"The contents of the first tumor is made up of yellowish liquid of an oily appearance, while the contents of the second tumor resembles a sebaceous cyst." He summarizes by saying that "The tumors contained two distinct elements; the one, oily, formed of a yellowish liquid which discharges readily when the capsule is cut — the other, much more solid, and resembles the contents of a wen. The patient made an uneventful recovery."

Arnold Knapp[8] reported a case (female, age 27) and gave a description and chemical analysis of the contents of the cyst. He mentioned that a firm mass was felt on palpation within the outer orbital margin. This was also present in my case. Microscopic examination was as follows: The sac wall was composed of thin connective tissue without any papillary projections. There was an inner lining of several layers of epithelial cells and keratin. The cyst wall contained very many sebaceous glands and a few hair follicles, and in the surrounding tissues were many dilated venules. The oil within the cyst contained no free fatty acid: 36.02 cholesterol, and iodine No. 124. He further states that "Oil cysts of the orbit belong to the group of dermoid cyst tumors. The proportion of oil contents depends upon the preponderance of sebaceous glands."

The case of oil cyst, which I am reporting, is interesting because of the fact that it was not noticed as early in life as the other cases that I have reported, and secondly that when examined microscopically it proved to be a carcinoma, grade 2, on basis of 1, 2, 3, 4.

Case Report

A Danish woman, age 53, consulted me because she had noticed that the left eye was becoming more prominent than the right and that she had pain in the outer upper angle of the left eye for the past ten days. She had also noticed that the upper part of the conjunctiva was red and inflamed. She did not complain of double vision.

Careful questioning of the husband elicited the fact that the left eye had been more prominent for the past ten years or more, neither of them had paid any attention to it, but that they were both certain that the eye had become much more prominent during the past three months.

Examination — The eye was proptosed downward and inward. The upper lid covered about one-third of the cornea. Digital examination revealed a bone-like mass wedged over the outer angle of the orbit. This immediately suggested to me the possibility of an osteoma of the orbit. The vessels in the upper portion of the conjunctiva were enlarged and the upper part of the conjunctiva looked inflamed. The cornea was clear, the pupil reacted to light and accommodation, the media were clear. There was about one diopter of swelling of

the nerve head. There was limitation of the movements of the eye outward and upward. The vision in the right eye was 20/30 and the left eye 20/40, when corrected with minus spheres. The anterior auricular, the submaxillary, and the submental glands were not palpable. X-ray examination failed to reveal any evidence of osteoma. A general physical examination was negative, except for marked obesity.

Operation — The patient was rather anxious not to take ether, and so I decided to attempt operation under local anesthesia. This I did without any complaint of pain. I had planned on doing the typical Kroenlein operation; after I made my incision and dissected the skin down to the orbital margin, I was able to remove the bony-like mass from the orbital rim and found it to consist of dense cartilage. There seemed to be considerable room, and instead of doing Kroenlein's operation, I was able, with Sewell's ethmoid retractor, to retract the eye sufficiently to allow me to dissect around the mass; however, as I dissected further back into the orbit, there was not room for me to continue without rupturing the cyst. This I did, and about a dram of dark oily fluid escaped. After the collapse of the cyst, I was able to dissect down to the apex of the orbit outside of the muscle funnel and remove what I thought was the entire cyst. After the removal of the cyst, the eye could be pressed back into the orbit very easily. A small rubber drain was inserted, the wound was closed, and a pressure bandage was applied.

The entire cyst was sent away for pathological diagnosis.

The patient had very little reaction, and the eye remained back in the orbit after the third day.

Pathology — The tissue is dense, hard, and cystic. Sections of the wall of the cystic tumor show no epithelial lining but consist of a dense, thick, hyaline tissue lined by a single layer of endothelium. In the cyst wall and deeper tissue, there is squamous cell carcinoma in masses, cords, and in acinous formation lying within the tissue and within lymphatic spaces. In addition to the carcinoma, there is much inflammation, chronic and subacute in nature, as evidenced by the presence of scattered collections of lymphocytes, few plasma cells and an occasional eosinophile and polymorphonuclear leucocyte.

Hemorrhage has taken place at some time which is shown by the presence of scattered blood pigment and characteristic "crystalline" spaces. The carcinoma is grade 2 (high).

Some of the hardness and denseness of the tissue might have been due to the rather strong solution of formaldehyde in which it was placed.

A week elapsed before I had received the above pathological report. In the meantime, the patient apparently had made an uneventful recovery with the exception that the anterior auricular glands were palpable. She refused to submit to an exenteration, and I advised her that she should receive radium therapy. The patient was finally given 23,900 mg. hrs. of radium over the left temporal, cervical and nasal regions, fourteen days after the operation. Following this she had a rather violent reaction which was characterized by edema of the lids and some nausea. This large amount of radium seemed to have had very little, if any, effect on the progress of the carcinoma. The eye again became proptosed. I informed the husband that her condition was hopeless. They put themselves in the hands of a cancer "quack," who removed the entire side of the face, including the nose and ear, with an arsenical paste. The patient died seven months later with a generalized carcinomatosis.

SUMMARY

I know now that dermoid oil cysts can become malignant. In the future, I will always have a pathologist at hand at the time of operation of any orbital tumor and have a frozen section made immediately, regardless of how benign it appears.

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DISCUSSION

Dr. Richard C. Gamble, Chicago: Dr. Jones' paper is of great interest to me because I have never seen or known of a similar case. My experience with dermoid cysts has been similar to that which you all have had, I am sure. One quite frequently sees soft oval subcutaneous swellings about the rim of the orbit, in children, which were present at birth and have later on enlarged to some extent. Usually, they are under the brow at either end, but may be above or below either canthus. The skin is movable freely over the mass, but the mass is often somewhat fixed to deeper structures, either because of attachment to the periosteum or because of extension of the cyst deeper into the orbit. Cysts of this superficial type are ordinarily removed for cosmetic reasons, while the child is quite young, and I have never found the contents to be oily to any great degree in children. It is true that sometimes the cyst contains a watery fluid in which are suspended flakes of cast-off epithelium, but this fluid is the product of the sweat glands present in the lining of the cyst. The usual content of such a cyst is an opaque yellow-white flaky substance composed of cast-off epithelium and a few hairs. On the other hand, I have seen similar superficial dermoids in adults which contained a large amount of oil. The least common type of dermoid cyst is the one which is entirely retrobulbar. This type also is present at birth, but its presence is usually unknown for many years. Finally, it enlarges sufficiently to produce the signs of a retrobulbar tumor. It is in this type that oil is most frequently found.

It would seem, therefore, that the production of oil in a dermoid cyst is the result of a very slow degenerative change. We know that changes do occur in these lesions. As the lining epithelium continues to secrete and to be cast off into the closed space the pressure may rise sufficiently to produce atrophy of some of the lining epithelium. If the sebaceous glands survive this pressure the best, the production of stearates and oleates may become dominant. Sometimes inflammatory changes occur which may directly decompose the opaque contents of the cyst into oil. When one considers these changes going on in an abnormal rest of epithelium, it is not surprising to find carcinoma developing in it; it is surprising that it has not been noted more frequently. We are, therefore, greatly indebted to Dr. Jones for bringing it to our attention.

Dr. Mary K. Asbury, Cincinnati: My remarks seem redundant after hearing the author's paper and Dr. Gamble's discussion. I hope you will bear with me.

The presence of oil cyst in the orbit, closely associated with squamous cell carcinoma, is best explained by supposing that the malignant changes occurred in the wall of an existing dermoid. I have found no similar case in the literature, but my search has not been exhaustive. From this point of view, Dr. Jones' case presents unusual features, but I believe they can be satisfactorily explained.

The majority of intraorbital dermoids are located between the globe and the outer orbital wall, in the pre-lacrimal region, and many described in the literature extend deep into the apex of the orbit. The tumor reported by Dr. Jones was so located.

The essential histological feature of the dermoid cyst is the epithelial lining. It may be well developed like epidermis with papillæ, but more commonly is rudimentary and often composed of only one or two layers of ill-formed epithelial cells.

The contents of the dermoid cyst is made up of the products of the epidermoid lining, that is, desquamated epithelial cells, sebaceous material, hair and sweat. If the desquamated cells predominate, the contents is described as atheromatous. A clear, oily content is a rare occurrence and is reported to be accompanied by the presence of numerous sebaceous glands in the cyst wall. The younger the individual, the more regular the arrangement of the epithelial constituents in the cyst wall.

As the secretion of the gland accumulates, the cyst becomes distended, the wall stretched and thin. The glands and hair follicles degenerate. Inflammatory changes occur in the walls of old cysts, resulting in the formation of masses of granulation tissue.

The pathologist's report describes the cyst wall as consisting of dense, hyaline tissue, lined with a single layer of endothelium. He said there was evidence of chronic and subacute inflammation in the cyst wall, in addition to squamous carcinoma.

The lack of a definite epithelial lining, glands and hair follicles in the cyst wall may be ascribed to the primary rudimentary form of these structures and to the degenerative changes which had occurred. A dermoid is a congenital cyst and therefore is potentially as old as the patient. This one was fifty-three, allowing ample time for degenerative changes to have occurred.

Parsons, in his textbook on eye pathology, says that in rare instances dermoids may be devoid of hair and sebaceous glands. Perhaps this case was one of these.

The author has referred to the case reported by Dr. Knapp in Archives of Ophthalmology in 1923. Histologically, Dr. Knapp's case did not resemble this one, but clinically, apparently, they were very similar. In Dr. Knapp's case, sebaceous glands were numerous in the thin cyst wall, but this patient was much younger than Dr. Jones' patient, and inflammatory changes, according to the pathological report, had not occurred.

According to Ewing, malignant changes in dermoids are not rare. Many varieties have been reported. Almost every recognized form of malignant tumor has been described. However, the most common form reported is squamous cell carcinoma, which is in accord with the epidermal origin of dermoid cyst. This one was graded *2* by the pathologist, that is, one of moderate malignancy, but with a tendency to metastasize.

Considering the short postoperative history and the early fatal outcome, one must conclude that the tumor was rather more malignant than the pathologist thought it was, but the treatment, with arsenical paste, may have hastened the fatal outcome.

If we consider the orbital tumor presented by Dr. Jones as primarily a dermoid, it presents two features, either of which is uncommon to dermoid cyst in any region, namely, the oily content and the carcinomatous metaplasia. Occurring simultaneously in the orbit, the tumor is rare indeed, and worthy of the careful study which it has been given.

THE RELATION OF STRABISMUS TO RIGHT OR LEFT SIDEDNESS

WALTER H. FINK, M.D.
AND
BRYNG. BRYNGELSON, Ph.D.
(By Invitation)

MINNEAPOLIS

AS IN THE PAST, the strabismus problem continues to be the subject of much discussion. Although many phases of it are well understood there still are some which are not. That we are not in possession of all the facts related to it is evidenced by our unsatisfactory therapeutic results in certain cases. In searching for the reason for this, we must conclude that our treatment is not based upon a thorough knowledge of the mechanism involved. It is true we are familiar with certain etiological factors, these being mostly related to the peripheral mechanism. However, we must admit the knowledge regarding the higher centers is still very meager. A better understanding of this central control, including its location, its method of functioning, and its relation to other higher cerebral processes, would facilitate greatly our knowledge of its dysfunctions and its rôle in the production of squint. We, as ophthalmologists, may be prone to look upon strabismus as a strictly localized condition related to the eye and forget that it may be but one phase of a generalized disorder. We must not err as did the early workers in speech pathology, who looked upon all speech defects as single, isolated conditions instead of a generalized incoördination of the entire mechanism involved in speech. As soon as this was recognized their cures were greatly augmented. Could it not be possible that certain cases of strabismus are dependent upon a dysfunction of the higher centers with associated dysfunction of other higher centers such as speech, writing, etc.? In other words, might it not be a manifestation of a generalized instability or poorly coördinated organism?

It seems logical that our future progress in this field will depend upon a better knowledge of the workings of the central

mechanism involved. With this thought in mind we have attempted to approach the problem of the etiology of strabismus by obtaining further insight into the functioning of the higher centers, stressing particularly the possible relation of the dysfunction of the fusion centers to that of other brain centers, especially the speech center. With the knowledge known to the speech pathologist that stuttering is but one manifestation of a generalized incoördination of the central nervous system, we have investigated the possibility that certain cases of strabismus may be another manifestation of the same condition.

Sixty unselected cases of convergent strabismus were analyzed and our paper is presented as a preliminary report of this work. We freely admit that the possible relationship between the factors causing strabismus and the dominant cerebral center controlling the sidedness of the individual may seem remote and it is possible our contribution will in no way throw even a ray of light upon the problem. We will, however, feel amply repaid for our efforts if our investigation will but stimulate discussion or perhaps induce others to do more work on this important subject.

Our interest in the problem was first aroused by a 5-year-old boy, who, while being treated for strabismus, began to stutter. The treatment consisted of using his less dominant hand in drawing. The right eye being convergent, the patient was instructed to work with his right hand and had been tracing pictures as used in the keystone set for several weeks, when the stuttering was first noticed. Dr. Bryngelson, the co-author, was consulted. Examination revealed a generalized lack of coördination. This apparently was precipitated by the change in sidedness. The patient was left-handed from birth and had remained so with the exception of using the right hand for eating. The addition of right-handed drawing was sufficient to disturb the dominant center for speech and handedness, and thus produce in the left cerebral hemisphere a competitive control over the midline levers of speech. When drawing with the right hand was discontinued, stuttering subsided. The possibility was suggested that the strabismus was another manifestation of this lack of coördination, or the competitive cerebral dominance, and that there may be a definite connection between the disturbance in speech and the strabismus. If a nervous system is so unstable that shifting to the use of the less dominant side will precipitate

stuttering, very likely such an unstable nerve control may precipitate strabismus.

To investigate this thought further, 60 cases of convergent strabismus were studied, both from the oculist's standpoint and from the point of view of the speech pathologist. Our objective was to determine what percentage of these cases showed evidence of lack of coördination of other parts of the body and whether the factors which precipitate incoördination, such as shifting the sidedness of the individual, etc., were present in these cases. The handedness and speech history of these patients was obtained by clinical examination and by personal interviews of the parents.

An analysis showed that practically 60% of the cases had been shifted from the dominant to the less dominant side. Also, that in the present use of their hands 41% + were ambidextrous; 58% + were using the right hand only. It was also determined that 74% of these patients had a family history of left handedness, i.e., they came from a left handed stock; 64% had a family history of twinning; twinning is often associated with left handedness; 18% + had a history of stuttering; 39.5% had speech defects, including stuttering; 21% had a family history of stuttering, and 62% had a family history of strabismus.

Reference to the refractive error, vision, degree of fusion, etc., is purposely omitted in the summary, because there is no direct relationship to the subject under discussion.

The following table is an analysis of the cases studied.

NOTE TO TABLE

Abbreviation	*Meaning*
S. S.	sound substitution
O. I.	oral inacuracy
M. and Mat.	maternal side of family
P. and Pat.	paternal side of family
Ambi.	ambidextrous in handedness
Stut.	stuttering
Sil. Reading	silent reading
Inc.	incomplete history
Sw.	sweeps
Sh.	shovels
Ra.	rakes
R.	right
L.	left
A.	ambidextrous
?	uncertain
No Inf.	no information
M.	male
F.	female
Bat.	batting preference
Bro.	brother
Sis.	sister
L. to R.	shift from left to right

NO.	SEX & AGE	GRADE	ONSET OF STRAB.	STRAB. EYE	REFRACTION	VISION	PRESENT HANDEDNESS	SHIFT	BIMANUAL	LEFT-HANDEDNESS IN FAMILY	TWINNING	HISTORY STUT. INDIVID.	HISTORY OF STRABISMUS IN FAMILY	HISTORY STUT. FAMILY	SPEECH DYS-FUNCTION
1	M-4	None	2½ yrs.	Right	+2.00 +.50×90 +2.00	6/30 6/6	Ambi.	Yes; L to R R to L	——	——	——	Yes	——	——	——
2	F-3½	None	1 yr.	Right	+6.50 +5.50	6/30 6/7	——	No	——	2 Pat. Cous. 1 Mat. Cous.	Yes 3 set Pat.	No	No	No	No
3	M-10	4-A	1½ yrs.	Left	+2.50 +.25×90 +3.00 +.50×90	6/6 6/9	Ambi.	Yes	Ambi.; sw. L. other R.	Mo.A;2 Mat.Aunt 1cs.M.Gr.M.A.	No	Yes	Yes Mother	No	Stut.
4	M-18	Sen.HS	5 yrs.	Left	+1.50—.25×60 +2.00—.25×75	6/6 6/6	Right	Yes	Ambi.	Pat. Gr. Unc. Mat. Gr. Aunt	Yes — M.	Yes	Yes Pat. Cous.	Yes Pat.Aunt	Stut.
5	F-7	2-A	3 yrs.	Left	+2.75 +.50×105 +3.00 +.75× 75	6/7? 6/60?	Rt.(ambi. bat. pref. Left)	No	Right	Pat. Unc. Ambi. Mat. Cous. L.H.	Yes — M.	No	No	No	No
6	M-7	2nd	4 yrs. br.L.arm	Left	+4.75 +1.00×90 +5.75 + .75×90	6/6 6/30	Ambi.	Yes	Right	Pat. Cous. L.H.	Yes — Pat.	No	Yes — Bro. Mat. at Unc.	No	O. I.
7	M-5	Kdng.	3 wks.	Right	+2.50 +.75× 45 Ret. +2.25 +.25×180 Ret.	6/60 6/6	——	No	Ambi. Sw. Sh. R. Ra. Ambi.	——	——	No	No	No	Articu-lation
8	F-8	3rd	Birth	Left	+1.50 +1.00×90 +2.25 + .50×90	6/6 6/12 Ret.	Right	No	Right	No	Yes — M.	Yes	Yes — Mo. Mo.Gr.Fath.	No	Slight stut.
9	F-5	Kdng.	3 yrs.	Left	+4.75 +.50×90 +5.50 +.75×90	6/9 6/12?	Right	No	Right	Mat. Gr. Aunt Pat.Unc.Mo.Ambi.	Yes — Pat.	No	Yes Mat. Aunt	No	No
10	M-8	3rd	2 yrs.	Right	+3.00 +1.00× 90 +4.00 + .50×120	6/60 6/6	Ambi.	Yes	Left	——	——	——	——	——	Articu-lation
11	M-12	8th	8 yrs.	Left	+2.00 +.25×90 Ret. +5.00 +.50×90 Ret.	6/5 6/61	Right bat. ambi.	?	Right bat. Ambi.	Yes—2 sisters	——	——	——	——	No
12	M-2	None	15 mos.	Right	—2.00 +1.50×90 —2.00 +1.50×90 Ret.		Right?	No	——	Pat. Unc. & Aunt M. & F. Ambi. Mat. Aunt L.H.	Yes — Mat.	——	Yes — Mat. Aunt L.H.	St. Fa. Ambi.	Retarded
13	F-12	7th	3 yrs.	Left	+3.00 +4.50	6/6 6/60	Right Bat. L'ed.	No	Rt. Ra. R. Sw. L.	No	Yes — Mat.	No	Yes Father	Yes Father	No
14	F-10	5th	3 yrs.	Left	+6.50 +2.00×90 +6.00 +1.75×90	6/9 3/60	Right	No	Right	No	Yes — Mat.	No	Yes Father	Yes Father	Sil. Read Spell.

NO.	SEX & AGE	GRADE	ONSET OF STRAB.	STRAB. EYE	REFRACTION	VISION	PRESENT HANDEDNESS	SHIFT	BIMANUAL	LEFT-HANDEDNESS IN FAMILY	TWINNING	HISTORY STUT. INDIVID.	HISTORY OF STRABISMUS IN FAMILY	HISTORY STUT. FAMILY	SPEECH DYS-FUNCTION
15	M-6	1st	1½ yrs.	Right	+1.00 + .50	6/6- 6/6	Ambi.	Yes R to L	——	Yes—Mat. Aunt L.H.	Yes — Mat. (b.g.rf.L.H. Aunt)	No	No	No	No
16	M-11	6th	——	Left	+2.50 +2.50	6/12 6/12	Right	Yes — 2	Right	Yes—2 sisters; 1 brother	Yes — Pat.	No	Yes Brother	No	No
17	F-8	3rd	2 yrs.	Right	+3.00 +2.75	6/12 6/15	Right	Yes	Right	Yes — M.	No	——	Yes — Sis. Brother	Yes Brother	——
18	F-4	None	1 yr.	Left	+4.00+ .50×75 +5.00+1.50×90?	Uncer-tain	——	——	——	——	——	——	——	——	——
19	F-6	1st	3 yrs.	Left	+5.75+1.75×90 +6.00+1.75×90	6/9 6/60	——	——	Right	Yes—M. & P.	No	Yes	No	No	Stut.
20	M-16	H.S.	3 yrs.	Right	+1.75 +1.75+.75×5	6/30 6/21	Right	Yes	Right	Yes Mother	No	Yes	Yes — M.	No	Wr. Sp. Read. Stut.
21	F-14	H.S.	5 yrs.	Left	+2.00+.50×90 +2.50+.37×75	6/15 6/15	Ambi.	Yes	Right	No	No	No	No	No	Sp. Sil. Read.
22	F-9	4th	1 yr.	Left	+7.25+1.00×25 +8.00+1.25× 5	6/6 6/60?	Ambi.	Yes — 6	Right	No	No	No	No	No	Spell.
23	M-11	6th	Birth	Right	+6.50+.75×180? +4.00+.25×105	6/60 6/6—1	Ambi.	Yes	Right	Yes Brother	No	No	No	Yes Sister	No
24	M-13	8th	——	Left	+2.00 +2.50+.25×100	6/6 6/30—1	Right	?	Left	——	Yes — M.	——	No	No	Write
25	F-10	5th	11 mos.	Right	+1.75 +1.50+.25×150	6/6 6/9	Ambi.	Yes	Left	Yes—Moth. 2 Sisters	Yes — P.	No	Yes—Mother Father	No	No
26	M-13	8th	——	Right	+3.00+1.50×90 +3.50+1.25×90	6/15 6/9	Right	No	Right	A — Father	——	Yes	Yes Mother	Yes — Mo. Fa. P. Unc.	Stut.
27	F-8	3rd	4 yrs.	Left	+4.25+.25×90 +4.00+.50×80	6/7 6/9	Right	Yes	——	Yes — Mother Brother	——	No	——	Yes M—Aunt	No
28	F-13	8th	4 yrs.	Left	+3.00+2.00×105 +2.50+1.50× 75	6/12 3/60	Right	No	Ambi.	——	No	No	No	——	No
29	M-6	1st	3 yrs.	Left	+5.50 +6.25+.50×30 Ret.	6/6 6/6	Ambi.	Yes	Ambi.	A — Father P. & Mother	Yes — P.	——	Yes — Pat. Cousin	——	——

NO.	SEX & AGE	GRADE	ONSET OF STRAB.	STRAB. EYE	REFRACTION	VISION	PRESENT HANDEDNESS	SHIFT	BIMANUAL	LEFT-HANDEDNESS IN FAMILY	TWINNING	HISTORY STUT. INDIVID.	HISTORY OF STRABISMUS IN FAMILY	HISTORY STUT. FAMILY	SPEECH DYS-FUNCTION
30	F-13	8th	4 yrs.	Left	+1.00+ .75×90 +1.25+1.25×93	6/6 6/6	Ambi.	Yes; Attempt at 6	——	Yes — M.	——	No	——	——	No
31	M-15	H.S.	6 yrs.	Rt.(out)	+2.25 +1.25+.25×90	6/30 6/6	Right	Yes	——	A — Father Pat. Uncle	——	Yes	Yes—Mother Brother	No	Stut.
32	M-7	2-A	2½ yrs.	Left	+2.25+1.00×90 +2.25+ .50×75		Right	No	Right	Yes — P. & M.	No	No	Yes Mother	No	No
33	F-23	None	(begin.)	Left	+.25+.25× 75 +.25+.62×120	6/7 6/7	Ambi.	Yes (begin.)	Left	Yes — Sister Mother A	No	No	No	No	No
34	M-4	None	6 mos.	Right	+5.00+3.00×105 Ret. +5.00+2.50× 75 Ret.	Uncertain	Right	Yes	——	Yes — Mother on Mat. Side	——	No	Yes — M.	No	No
35	M-15	Frosh.	13 mos.	Left	+1.50+ .50× 90 +2.25+2.00×150	6/6 6/30	Ambi.	Yes	——	——	——	——	——	——	——
36	M-4½	None	3 yrs.	Right	+4.00+3.00×16 +3.75+ .25×90	6/60 6/7	Right	No	Right	Yes Twin Sister	Yes M. & P.	No	No	No	No
37	F-23	None	1½ yrs.	Right	+3.25 +3.00+.25×90	6/12 6/7	Ambi.	Yes	——	No	No	No	Yes Mother	No	——
38	M-1½	None	1½ yrs.	Left	+3.00 +3.00	6/6 6/30	Ambi.	Yes	——	Yes — P.	Yes — P.	——	No	——	——
39	F-8	3rd	4 yrs.	Left	+2.00+.87×110 + .25+.62× 72	6/6 6/12	Ambi.	Yes	Amb.Ra.L. Sw.Sh.R.	Pat. Cous. A. Father A.	Yes — M.	No	Pat.cs.etc. Mat. side	No	Lisp
40	M-32	None	3 yrs.	Left	+2.75+.50×90 +3.50+.25×90	6/6 6/12	Right	?	Right	Pat. cous. & his son L.H.	Yes — M.	No	Yes — Sister Father	No	No
41	F-7	2-A	8 mos.	Right	+3.00+1.00×75 +3.00+1.00×90	6/60 6/6	Ambi.	Yes	Left	Father A. Mother L.	Pt. twin 1 set P.	Yes 4 m.	Yes — Cousin gr. father	No	Stut.
42	F-9	4-B	2 yrs.	Left	— .25+ .25×135 —1.25+2.00× 90	6/6 6/30	Right	Yes	Right (a ocu.)	Yes Sister	Gr.Aunt twin on both M.&P.	No	Yes — Pat. Cousin	No	Nervous Speech
43	M-3	None	2½ yrs.	Right	+1.50 +1.50	6/7 6/9	Right	No	——	——	——	——	——	——	——
44	F-24	None	4 yrs.	Right	+4.75+1.50×60 + .75+ .50×90	6/60 6/6	Ambi.	Yes	Left	Yes — Pat. Aunt 2 Cousins	Yes Pat.	No	No	No	No
45	M-5	None	——	Right	+2.25 +2.50+.25×105	Uncertain	Ambi.	Yes	——	——	——	——	——	——	S. S.

NO.	SEX & AGE	GRADE	ONSET OF STRAB.	STRAB. EYE	REFRACTION	VISION	PRESENT HANDEDNESS	SHIFT	BIMANUAL	LEFT-HANDEDNESS IN FAMILY	TWINNING	HISTORY STUT. INDIVID.	HISTORY OF STRABISMUS IN FAMILY	HISTORY STUT. FAMILY	SPEECH DYS-FUNCTION
46	M-10	5-B	4 yrs.	Right	+6.00 +5.00	3/60 6/9	Right	No	Right	No	———	No	——	——	No
47	F-14	H.S.	——	Left	+4.00+1.00×90 +5.00+1.00×90	6/6 6/30	No	——	Right	———	No	No	No	——	No
48	M-7	2nd	Infancy	Right	+3.00+.75×115 +2.75	6/60 6/12	Right	No	Right	Yes — M.	No	No	No	No	No
49	F-17	H.S.	Infancy	Left	+2.75+.50×105 +2.50+.50×120	6/6—2 6/12	Right	No	Right	No	Yes — Pat.	No	No	Yes Bro.	No
50	M-7	2nd	1½ yrs.	Left	+5.50+.25×80 +5.50+.25×75		Right	?	Right	Yes Brother	———	No	——	——	No
51	M-5	Kdgn.	Birth	Left	+2.75+1.00× 90 +1.50+1.25×135	Left fixation lost	Ambi.	Yes	Right	A — Father Brother	No	No	Yes Brother	——	No
52	M-8½	3rd	Birth	Left	+3.25+ .75× 80 +4.00+1.00×105	Left cent. fixa. lost	Ambi.	Yes	Right	A — Father Brother	No	No	Yes Brother	——	No
53	M-11	6th	3 yrs.	Right	+5.50+ .50×90 +5.00+1.00×90	6/20 6/9	Ambi.	Yes	Left	Yes — Sister Pat. Uncle	No	No	No	No	Spelling
54	F-5	Kdgn	2 yrs.	Right	+8.00+.25×180 +8.25+.25×180	6/60 6/60	Right?	——	——	Yes Brother	Yes 2 sets M.	No	——	——	No
55	F-11	6th	2 yrs.	Left	+5.00+3.50×105 +4.50+3.50× 80	6/9 6/12	Right	No	Ambi.	No	Yes — M.	No	Yes—2 sis. 2 brothers	No	No
56	F-9	4th	——	Left	+4.75+2.00×90 +5.50+1.00×75	6/21+1 6/50+1	Right	No	Ambi.	No	Yes — M.	No	Yes	No	No
57	F-8	3rd	——	Left	+4.00+1.00×75 +5.00+ .50×90	6/9 6/15	Right	No	Right	No	Yes — M.	No	Yes	No	No
58	M-6	1st	1 yr.	Left	+4.75 +4.75	6/6 6/12	Right	No	Ambi.	No	Yes — M.	No	Yes	No	No
59	M-4	None	1 yr.	Left	+7.50+1.50×90 +8.00+1.50×75	6/12 6/60	Right	No	Right	No	Yes — M.	No	Yes	No	No
60	M-8	3rd	4 yrs.	Left	+5.75 +5.25+.75×85?	6/9 6/60?	Ambi.	Yes	Right	Yes — 1 Bro. 2 Sisters	Yes — Pat.	No	Yes Brother	No	No

Summary of 60 Cases

Sex — Male 32 or 55%. Female 28 or 45%. Ages 2 yrs. to 32 yrs.

Strabismus — 60 cases.

Right eye ..... 37 or 61.66%
Left eye ..... 23 or 38.33%

Present Handedness — 55 cases gave definite information.

Ambidextrous ..... 23 cases or 41.82%
Right Handed ..... 32 cases or 58.18%
Left Handed ..... No cases

Shift of Handedness — 52 cases gave definite information.

Yes ..... 31 or 59.62%
No ..... 21 or 40.38%

Left Handedness in Family — 50 cases gave information.

Yes ..... 37 or 74%
No ..... 13 or 26%

Twinning — 45 cases gave definite information. (This information went back as far as grandparents.)

Yes ..... 29 or 64.44%
No ..... 16 or 35.55%

Stuttering in Individual — 49 cases gave definite information.

Yes ..... 9 or 18.39%
No ..... 40 or 81.63%

History of Stuttering in Family — 43 cases gave definite information.

Yes ..... 9 or 20.93%
No ..... 34 or 79.07%

Speech Defects Including Stuttering — 53 gave definite information.

Yes ..... 21 or 39.62%
No ..... 32 or 60.37%

History of Strabismus in Family — 48 cases gave information.

Yes ..... 30 or 62.50%
No ..... 18 or 37.50%

This analysis emphasizes chiefly the high percentage of cases shifted from one side to the other. The fact that the shifts were made early in life, usually before the onset of the strabismus, would seem to be significant. The high percentage of speech defects associated with them is also significant, and the fact that these cases appear to come from a left-handed family stock.

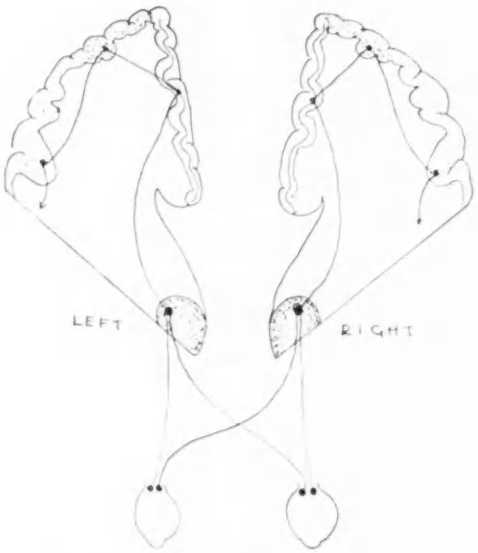
To explain the possible relationship between the occurrence of strabismus, speech defects, and the effect of shifting of sidedness, one must be familiar with the functioning of the higher centers of the brain. Most of our knowledge in this field we owe to the research of the speech pathologist. Unfortunately, the ophthalmologist has not delved into the realm of brain physiology, but, instead, has devoted his efforts to the understanding of the peripheral mechanism. The speech pathologist, however, has for several years been attempting to explain speech disturbances from the higher control angle and consequently it is he to whom we must turn for knowledge. His researches in the field of neuro-physiological mechanisms of the brain have been extensive and much evidence has been collected as to the basic processes involved. His theories are sound and many of them have been substantiated by the experimental work of Orton, Lashley, Child, Coghill, and Weisenburg.

That a similar situation exists in the realm of ocular imbalance as in speech dysfunction is possible. In both conditions, we may have a similar basis and the speech pathologist's line of reasoning may apply to our problem. Briefly stated, the speech pathologist believes that the cerebral cortex is the seat of control over the lower centers through which the peripheral mechanism is controlled. The control is not sponsored by a definite isolated center, but, rather, by a diffuse association area, which is intimately linked with other association areas. For perfectly executed speech, one cerebral hemisphere must dominate the other. If it does not, both hemispheres are equally active and incoördination of the bilaterally-innervated peripheral structures results; speech defects being but one phase of the picture. Changing from the use of a dominant hand to the use of the less dominant hand may precipitate incoördination of higher cerebral processes by activating the less dominant hemisphere, and forcing it to function antagonistically to the other. Trauma, febrile disease, injury to the brain may have a similar effect. Certain people are more predisposed than others to develop this difficulty, because their higher association areas are not complete in their maturation for strict one-sidedness. If nothing is done to upset this feebly developed balance the individual may go through life never suffering a disability in the highly integrated complex motor skills. If there is a lack of a strong neurophysiological bias for the development of cerebral dominance, incoördination in the peripheral musculatures may result.

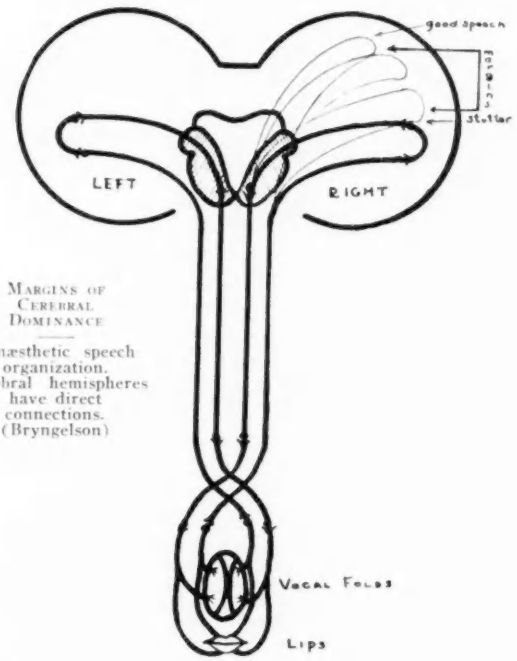
This line of reasoning may also be applied to the development of strabismus. If, in these individuals who do not possess a strong neurological bias for strict one-sided development, a sudden shock, a shifting of brain dominance, a severe illness, or an injury, may precipitate incoördination of function which may be generalized or may be localized in the ocular association area. That there is a unilateral hemisphere dominance in the ocular field seems probable and experiments indicate that the dominant side corresponds 75% of the time with the dominant areas controlling other functions such as speech, handedness, and footedness.

Conclusion

This paper is presented as a preliminary report. The number of cases analyzed does not permit definite conclusions. They do, however, suggest a probable relationship between strabismus and the inherent sidedness of the individual. It is possible that strabismus in certain cases may be like stuttering, another manifestation of a generalized disintegration of the higher neural processes and found in individuals who do not possess an adequate maturation of the higher association areas essential for the smooth functioning of sight.



Bilateral Nervous Organization of the Eyes



Margins of Cerebral Dominance

Kinæsthetic speech organization. Cerebral hemispheres have direct connections. (Bryngelson)

DISCUSSION

Dr. Bryng. Bryngelson, Minneapolis: In order to clarify the problem of the possible relationship of sidedness and strabismus, I would like to indicate what we in speech pathology have noticed about eye movements when photographed in stuttering.

Quite often in the height of a tonic spasm the patients experience binocular incoördination. This incoördination may be either of a vertical nature or of an horizontal type. One eye may remain fixed during a stuttering spasm while the other eye moves markedly up and down. Often horizontal incoördination is characterized by either a complete independence of movement of both eyes or an extreme convergent strabismus may be manifested.

Speech pathologists interpret the stuttering act as being representative of a type of nervous incoördination which is sponsored by the lower levels of the brain. In stuttering there is a lack of cortical inhibition over the lower centers of brain activity. This lack we feel is due to a breakdown of the unilaterality of nervous function. We think of one-sidedness as a symptom of this unilaterality. A stutterer represents an ambilateral condition in the central mechanism of the brain. Both cerebral hemispheres operate independently in the control of the bilateral peripheral mechanism of speech. Normal movement of bilateral peripheral structures requires a synchronization of nervous impulses, which can only obtain under the dominance of motor leads from one cerebral hemisphere. The shifting of native sidedness by altering handedness, and febrile disease, trauma, or accident, can cause the disintegration we see in stuttering.

May I also call your attention to the work of Diefendorf and Dodge relative to eye movements observed in certain insanities, wherein the activities of the higher centers are lessened. They noted in maniacal patients that the eye movements were not those requiring conscious direction and control. The slow movements found in depressed and epileptic patients, they thought, must be due to an involvement of the lower centers. Other types of pathologies revealed curious inconsequential fixations, inaccurate eye movements, the normal movements breaking at irregular points.

It is also a common observation that when the higher centers are decreased in activity during intoxication, the eyes do not track. We speak often of this condition as being "cock-eyed."

If in the future study of our cases there appears to be a decided relationship between sidedness and squint we would be prone to explain the phenomenon of squint as being one in which the higher cortical areas, which have to do with visual fusion, are disturbed—thus giving the function of coördination of eye movements to the lower levels. This condition might, then, well be obtained when the native sidedness of a child is confused. Strabismus in some cases might be another of many symptoms of a disoriented nervous system in which a dominant center for fusion is not allowed to mobilize its nervous energy in a manner which signifies a dominance of the higher over the lower centers of neural activity.

This study is merely a beginning of a larger study of 200 cases. What the future of this type of research will bring forth no one is able to say.

700 Stutterers; 60 Strabismus; 4000 General Population

Ambidexterity		*Shifted R. L.*	
Stutterers	61%	Stutterers	73%
Strabismus	42%	Strabismus	60%
General Population	5%	General Population	8%
L. H. in Family		*History of Stuttering in Family*	
Stutterers	83%	Stutterers	75%
Strabismus	74%	Strabismus	*21%
General Population	30%	General Population	8%
Speech Defects		*Stuttering*	
Strabismus	22%	Strabismus	18%
General Population	10%	General Population	1%

*62% Strabismus History in Family

Dr. Derrick T. Vail, Jr., Cincinnati: We are indebted to the essayists for venturing a new approach to the incompletely understood problem of strabismus. We have all seen among our squint patients children who have been nervous, irritable, shy and inadequate to meet their daily problems. All of us have also had the gratification of seeing a few of these recover the normal balance of behavior after the successful handling of their strabismus. The authors of this paper bring in a new angle. Instead of assuming that squint leads to behavior problems, they have, tentatively, and I might say even hesitantly, advanced the idea that poorly developed neuron association tracts have produced the squint. I don't believe they are too sure of their ground and certainly the discussant is not of his.

The subject of left handedness has not received, until fairly recently at least, the attention it deserves. One is not yet certain, for example, just what is the percentage of left handedness. Quinan found that 19% of over 2,000 university students were left handed. Ireland found about 12%. When we consider the importance of the master eye or ocular dominance, family history, early training and other factors, the rough classification into right and left handers must be further modified. Wile classifies the difficulties that are most common amongst left handed children who present behavior disorders into five separate groups. According to this writer there is a general group of difficulties including awkwardness, poor muscular coördination, irritability, restlessness, slow thinking and readily induced confusion. Among active special symptoms are slow speech, stuttering and mirror writing. He then goes on to different separate groups. Whoever is interested in the subject will read his article with profit. The point I wish to make is that psychiatrists are still at sea regarding left handedness. One will find authorities to support the theory that the left side of the brain is usually the better developed because of

its better nutritive supply through the arrangements of the vessels at the aortic arch and the larger caliber of the left carotid and thus the human race is predominantly right handed (Church and Peterson). And there are authorities (Tilney) to deny this. Watson, who has done much original work on this subject, believes in the old theory that our ancestors in ages past have learned by sad experience to cover the heart with a shield held in the left hand and thus use the right for jabbing or thrusting swords and spears. In other words, handedness is a habit socially instilled. We agree that there is apt to be a family history of left handedness in many cases. There are also abundant case reports showing that stuttering and other speech defects have followed shifting the individual from the dominant to the less dominant side.

Whether, however, such shifting has resulted in speech defects, *per se*, or whether the child has been made aware of a physical handicap in being left handed in a right handed world, thus leading to speech defects through inferiority, is a point which I think has not been satisfactorily settled. I think the essayists are attempting to put the cart before the horse. Certainly most of the strabismus cases have developed their squints before they learn to talk. Nor would it be remarkable to find at least 19% of squint cases left handed, if this is the correct percentage for the population as a whole. Again one could argue by saying that because of the squint, plus the left handedness, a child has a double defect which would affect his personality and lead to speech disorders on changing his dominant sidedness.

I don't believe, from the evidence the essayists have presented, even tentatively, that they have proved their premise. In the first place, 17 of 26 of their cases, which were shifted, were hypermetropic by at least 2 diopters in the better eye. The part hypermetropia plays in strabismus does not need to be discussed.

Again there is a certain confusion in my mind about terms. What determines shifting from a dominant to a less dominant side? Watson believes, from careful experimental work, that infants from the age of 150 days to one year show no steady or uniform handedness. How did the authors decide which hand was originally dominant? What would a similar group of non-squinting children of the same age show in regard to shifting? I may say in justice to the essayists, I didn't receive any communication from Dr. Bryngelson, so I had no idea he was to show those statistics. However, the question still stands. If infants up to one year are ambidextrous in the sense that they use either hand indiscriminately, does ambidexterity in the older child imply the same thing? Especially for finer movements, like writing and drawing?

When it comes to speech defects, there does not seem to be a common meeting ground for the speech pathologists. I should like to ask Dr. Bryngelson, who has especially studied this subject, what is the modern consensus of opinion regarding left handedness and stuttering? Does he subscribe to Watson's belief that "changing over a left handed, talking child suddenly into a right handed child is likely to reduce the child to the level of a six-months-old infant?" In other words, he means that "by interfering constantly with his acts we break down his manual habits

and simultaneously interfere with speech (since the word and the manual act are simultaneously conditioned)." If so, then strabismus exercises would be an added burden in such a case.

Is it likely that the high percentage of speech defects in Dr. Fink's series of cases (18.39%) might also be due, in part at least, to family language difficulties? By this I mean the influence of foreign, immigrant parents who do not speak English. Bilingualism can not be conducive to fluency of speech in either tongue.

Finally, I should like to know what grounds they have for believing that there is a unilateral hemisphere dominance in the ocular field.

The following tables are analyses of the authors' figures as well as an analysis of 142 squint cases occurring in the discussant's private practice.

TABLE I — *Fink and Bryngelson*
Analysis of 8 Cases Under 2D Refractive Error

6 had vision of 6/12 or better in each eye.
1 had vision of 6/6 in non-squinting eye.
1 had vision of worse than 6/12 in better eye.
3 cases had onset of strabismus before 2 years of age.
2 of these showed no speech defect (1 not mentioned).
4 cases had onset of strabismus between 3 to 5 years (1 case no age given for onset of strabismus).
¾ of these cases had speech defects.
2 of these 3 cases had family history of squint.
0 cases were left handed, 3 ambidextrous.
Leaving 1 case of non-refractive error squint and speech defect without family history of squint.

TABLE II — *Fink and Bryngelson*
Refractive Error in the Shifted Cases (31) — All Cases Cited Were Hypermetropic

8 or 2.6%	had refractive error of	Less 1D to 2D in better eye
5 or 1.6%	had refractive error of	2D to 3D in better eye
5 or 1.6%	had refractive error of	3D to 4D in better eye
4 or 1.3%	had refractive error of	4D to 5D in better eye
4 or 1.3%	had refractive error of	5D to 8D in better eye
5 or 1.6%	refractive error not mentioned	

17 out of 26 cases had refractive error over 2D in better eye.

TABLE III — *Fink and Bryngelson*

73.7 % of cases	Had onset of strabismus before age of 3
50. % of cases	Had onset of strabismus before age of 2
18.39% of cases	Had speech defect
39.62% of cases	Had family history of speech defect
59.62% of cases	Shifted from dominant to less dominant side

TABLE IV — *Vail*
142 Cases of Strabismus

43 Alternating Convergent Strabismus	30.2%
19 Alternating Divergent Strabismus	13.4%
58 Convergent Strabismus	40.9%
20 Divergent Strabismus	14.0%
2 Hypertropia	1.4%

TABLE V — *Vail*

16 originally left handed patients	11.2%
9 changed from left to right	6.3%
7 unchanged	4.9%
1 of those changed had a speech defect	
2 of those unchanged had a speech defect	
2 of the right handed had a speech defect	

TABLE VI — *Vail*

Analysis of Cases (5/142) of Squint Plus Speech Defect
1 case alternating divergent squint.
3 cases left divergent squint.
1 case alternating convergent squint.
2 cases left handed, unchanged.
2 cases right handed, unchanged.
1 case left handed, changed.
(This case had spinal meningitis at age of 5 with onset of left divergent squint, amblyopia and speech defect).
4 cases had squint since birth.

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DR. OSCAR WILKINSON, Washington: There are numerous very interesting phases of this interesting paper by Dr. Fink, but there is only one phase of it which I shall undertake to discuss at all, and that is the general neuro-instability that is observed, I would say, in 75% to 80% of cases of squint.

This I brought out in a little monograph I published a few years ago in reviewing the etiology of squint. I believe we have not been impressed by the importance of the neurological relation of squint. Of course, the muscular etiology of squint predominated for a number of years after the tenotomy was devised. Then Javal and Worth and others began to look at squint from a different angle. Finally, Worth came out with his so-called fusion theory.

If we will examine our cases of squint carefully from a neurological standpoint, I think we will find that a high percentage have a very decided nervous instability.

DR. EDWARD JACKSON, Denver: The two authors of this paper and Dr. Vail, in opening the discussion, made it pretty clear to us that they hadn't settled anything, didn't think they had settled anything, but they had opened up to us a new point of view with reference to strabismus. That, perhaps, is the very important thing that has been brought out.

Collins, twelve or thirteen years ago, pointed out the development of binocular vision and of accurate movements with the hands as occurring in our arboreal ancestors, among the early primates, among the monkeys. The development of binocular vision has been very definitely traced through the studies in evolution.

Sir John Parsons, in the 1925 Bowman lecture, took up a different phase of the evolution, different kinds of vision. Then Professor Elliott Smith, in 1928, in his Bowman lecture, took up a broader view of connecting the evolution of macular vision with the evolution of what he calls the neopallium, that is, practically, the cerebral cortex, the vast mass of the cerebral cortex, also, has been developed along with macular vision, binocular vision, binocular movements, and squint is a defect of binocular movement.

It is very well established in the annals of evolution that recently, relatively recently acquired things are more likely to vary, and binocular vision is imperfect, binocular coördination is imperfect on that account.

That is the general point of view that I think we ought to accept. Along with high hyperopia, along with what is often overlooked, temporary paralysis or paresis of some particular muscle in early childhood, along with those causes that are established as causes of squint, we must consider this broader coördination that is at least suggested by the coördination in time of development of the higher cerebral centers and the binocular movements.

Just one instance of that may illustrate something about the connection of right handedness and left handedness with vision. My father was distinctly left handed throughout his life in all the things he did. He acquired new movements more readily with his left hand and used his left hand, but always wrote with his right hand.

When I got into ophthalmic practice and began operating, I found that it was very easy for me to be ambidextrous for cataract operations, for instance. I operated on the right eye with the knife in my right hand, and on the left eye with the knife in the left hand. My daughter learned to write with her left hand. When she went to boarding school, where she passed her high school years, four years, she was very carefully and persistently instructed in writing with her right hand. She continued to write with her right hand until she went to college, and then she went back to writing with her left hand and continued to use that for writing to the end of her life.

I had a brother, an older brother, who used to amuse himself by writing backhand with his left hand. It is a very large subject, but I mention this family instance as illustrating the different things we have to consider and study, study in individual cases and study statistically, before we can have a comprehensive idea of the etiology of squint.

DR. SANFORD R. GIFFORD, Chicago: I like to see a good argument. I think in our American societies we are a little too apt to overstress politeness to the detriment of our scientific discussions, but I did think before Dr. Jackson got up that I wouldn't like to see Dr. Fink's paper washed up entirely by such an efficient discussion as Dr. Vail had given it.

When anybody goes into a new subject like this, they go into it with enthusiasm; otherwise, they wouldn't go into it. I think Dr. Fink and Dr. Bryngelson have expressed their aims very modestly, and that they have gone into it with an open mind. They are probably like all such people who start such pieces of work, over-enthusiastic about it. I think we will find, when their work has been done and other work has come along, that not a very large percentage, perhaps, but a small percentage of cases of squint are due to some such incoördination as they have described.

I have been interested in seeing some cases with a teacher here, the principal of a school, Dr. Farwell, who is interested in the problem of reading defects in children and has tried to find out whether this dominance has anything to do with it. She hasn't arrived at any very great conclusion, but she has tried to test the reading efficiency of these children when using one eye and then the other eye, and she had some rather interesting histories.

She is also trying in some cases, for instance, which have cross-downwardness — they have a dominant right eye and a dominant right hand, and a dominant left eye and a dominant left hand. She has tried to see, by covering the left eye, if the right eye would become dominant. It is an interesting possibility. I haven't seen that it would apply to many cases, but I do think we have to know about these cases, because teachers are learning about this, and we ought at least to know as much as they do about right and left sidedness.

DR. GEORGE P. GUIBOR, Chicago: For the last year and a half it has occurred to me that I might be producing a disturbance in attempting to overcome squint, so I have been studying the problem of whether stuttering is produced by fusion training. I have noticed that patients who stutter also have some squint at times.

There are several things that appear in the paper to obviate the findings. For example, 60% of these cases had a family history of squint; 62%, in fact. We know that there must be in many cases a family history of squint. The types of squint, the etiologic factors, have not been gone into in detail. For example, in my own superficial survey of a number of cases which have had defects in speech and writing, I have noticed that many of these, possibly 35%, are left handed and right eyed; that is, with speech defects and writing defects, but I have found up to

the present time no relationship between speech defects being produced by fusion training or writing defects.

So I should like the authors to tell us the test that they use for handedness, because my own son throws a ball with his left hand, and he eats with his right hand. On the other hand, I think in the handed tests, for example, the ball-throwing test or the revolver test, whatever test one would use, he would be left-handed. I know he is not ambidextrous.

I myself at one time attempted to train my left hand so I would become ambidextrous, and I started to stutter, so that I stopped that, but I didn't get any squint.

I think these facts should be clarified, especially the tests that were used for handedness and for eyedness.

Dr. Walter H. Fink, closing: I wish to assure you that I appreciate very much the discussion, particularly Dr. Vail's, although I personally feel it is rather devastating, considering the purpose of this paper. I fully realized in taking up a subject of this sort the possible criticisms which may come, but I agree perfectly with Dr. Wilkinson in saying that at least we must be courageous enough to approach a subject from a new angle, to see if we can uncover certain phases which up to this time are not known. The first consideration, relationship, seems somewhat perhaps uncertain, and no relationship at all.

Dr. Bryngelson and I do not for a moment pretend to have come to conclusions, as was suggested by one of the discussers. We merely wished to introduce the subject and, if possible, arouse criticism and thought. Personally, I feel that the question of strabismus, although we have solved many problems, is still somewhat unsettled in certain cases, be they isolated or more frequent. I feel that if we can uncover other features of this situation, it would be worth at least a discussion. The percentage is small, I agree, but we intend to do more work. I am not over-enthusiastic on the subject, and I am trying to present it for what it is worth, hoping that by so doing it will at least stimulate some discussion.

I don't for a minute wish to give the impression that the cases of this type are not hyperopic and that there are not some fallacies, but the main point I want to bring here is the fact that the speech pathologist has solved many of his problems by this explanation, and we, as ophthalmologists should at least recognize this fact and try to correlate our problem with his.

Contrary to Dr. Vail's statement, the speech pathologists have some definite findings on this subject, and they have come to very definite conclusions, particularly Travis of Iowa City, who is supposed to be one of the foremost authorities.

I have written to various men in this particular field, and they all gave evidence of interest, saying that nothing has been done on the subject and that it was well worth trying.

I hope you will keep in mind the fact that it is merely a preliminary report and that no conclusions can be given. We present the facts as they are, hoping that others will continue to work along these lines.

THE FUNDUS OCULI IN DIABETES MELLITUS*

M. L. Folk, M.D.

AND

Samuel Soskin, M.D.
(By Invitation)

CHICAGO

Although the medical literature is replete with articles on this subject, the numerous conflicting views regarding the frequency and causation of retinitis, as well as the status of the retinal vessels in diabetics, justify further intensive study of this problem.

Our report differs from most previous articles on this subject in that it is not a compilation of data obtained from routine case records. All observations here recorded, both ophthalmologic and metabolic, were made by the writers for the express purpose of this study. We believe that this procedure has rendered our data more accurate and more comparable than if the more usual method had been followed.

Our method of study was as follows: Diabetic patients were picked at random from the metabolic clinic of the out-patient department of the Michael Reese Hospital. Their fundi were studied minutely, with the pupils dilated, and the findings and diagnoses entered on special record sheets. As many of these patients as possible were reëxamined a second and, sometimes, a third time. When a total of 150 diabetic patients had been examined in this manner, a systematic examination and review of the diabetic status was carried out and similarly recorded. Since only 100 of these patients were sufficiently regular in their attendance to enable us to make an accurate estimate of their diabetic condition, our first two tables are based on this number of patients. However, the percentage incidence of retinitis and retinal sclerosis was approximately the same for

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the entire group of 150 as for the smaller group of 100. As a control measure, 150 non-diabetics, in approximately the same age groups as the diabetic patients, were drawn from the eye clinic and studied in a similar manner.

Before proceeding with the presentation of our data, the following criteria, adopted by us as essential for the differentiation of the various fundus conditions must be stated.

A. *Criteria for Diabetic Retinitis:*

1. A normal or nearly normal disc.
2. Punctate hemorrhages and small bright exudates in the macular region. This condition was first described by Hirschberg[1] in 1890 and later confirmed by Leber,[2] Dimmer,[3] and others.
3. Mild or no sclerotic changes in the retinal vessels.
4. Sharply defined, yellowish, soapy-looking plaques about the macula and elsewhere in the fundus.

B. *Criteria for Arteriosclerotic Retinitis:*

1. Disc usually waxy in color and poorly outlined, but not definitely edematous.
2. Fairly marked sclerotic changes in the retinal vessels — to be discussed later.
3. Numerous flame-shaped hemorrhages and woolly exudates scattered through the fundus, but no macular star.

C. *Criteria for Albuminuric Retinitis:*

1. Stellate or suggestion of stellate figure in macula.
2. Edematous discs and zone of edema around them.
3. Fairly marked sclerotic changes in the retinal vessels.

D. *Criteria for Sclerosis of Retinal Vessels:*

This condition is very difficult to evaluate, as so many signs have been mentioned by various observers, especially Gunn,[4] Foster Moore,[5] de Schweinitz,[6] Friedenwald,[7] Pinas,[8] etc. Some of these signs are found in normal retinæ, as congenital anomalies. However, we considered the retinal vessels as moderately sclerosed only when several signs were present, especially the following:

1. Moderate arteriovenous constriction at crossing of artery and vein.
2. Deflection of vein at point of crossing.
3. Poor visibility of vessel walls.
4. Fine white lines at crossings.
5. Silver-wire arteries.

Tortuosity of vessels, prominent central streak, and slight narrowing of vessels were not considered pathognomonic, unless found in addition to the above signs, for they are frequently observed in hyperopes.

In advanced sclerosis, in addition to the above signs, the following were found: (1) Beading, or localized constriction of the arteries. (2) Silver-wire arteries. (3) Very marked arteriovenous constriction at crossing; and (4) Perivasculitis, or white lines along vessel walls.

Guist's sign, namely, marked tortuosity of the macular venules, was found only a few times in our series and was, therefore, disregarded.

Observations

Tables I, II, and III illustrate our findings. Table I includes all of the 100 diabetic cases in which our data is complete, both those having a single fundus condition and those presenting a combination of pathologic fundus conditions. The distribution of single and combined cases is recorded under the corresponding headings in the column entitled "Number of Cases." Table II comprises cases of more than one pathologic condition in the fundus. It will be seen that this group together with the group of "single" cases in Table I, make up a total of 100 cases. Table III gives the findings in the control group of non-diabetics.

It may be seen from Tables I and II that the incidence of retinitis of all types was 33%, of central punctate retinitis alone 15%, and of combinations of different types 27%. Arteriosclerotic retinitis alone was found in 6% of the cases and in combination with other types of retinitis, in 14% of the cases. In addition to the above, we found one case of thrombosis of the central vein in a case of arteriosclerotic retinitis; also, one case of choroiditis. Extensive retinal hemorrhages were very rare and were seen only in the arteriosclerotic type of retinitis. As can be seen from Table III, the total incidence of retinitis in the non-diabetic group was only 6%.

TABLE I — *Diabetic Group — All Cases*

DIAGNOSIS	NUMBER OF CASES TOTAL	NUMBER OF CASES SINGLE FUNDUS CONDITION	AVERAGE AGE YEARS	SEX MALE	SEX FEMALE	AVERAGE DURATION OF DIABETES YEARS	SEVERITY OF DIABETES MILD	SEVERITY OF DIABETES MODERATE	SEVERITY OF DIABETES SEVERE	AVERAGE BLOOD PRESSURE SYSTOLIC	AVERAGE BLOOD PRESSURE DIASTOLIC	CARDIO-VASCULAR RENAL PATHOLOGY PRESENT	CARDIO-VASCULAR RENAL PATHOLOGY ABSENT	COOPERATION OF PATIENT IN TREATMENT GOOD	COOPERATION OF PATIENT IN TREATMENT FAIR	COOPERATION OF PATIENT IN TREATMENT POOR
1. Diabetic Retinitis	27	15	55	7	20	9½	7	19	1	166	95	13	12	8	7	12
2. Non-Diabetic Retinitis																
A. Arteriosclerotic	14	6	57	5	9	9	5	8	1	171	96	10	4	4	7	3
B. Albuminuric	3	0	57	1	2	7	1	2	0	150	86	1	2	1	0	2
3. Sclerosis of Retinal Vessels																
A. Moderate	29	28	54	12	18	6	12	15	3	139	83	8	21	10	7	13
B. Severe	5	5	59	1	4	5	4	1	0	166	95	2	3	2	1	2
4. Negative	34	34	49	14	20	7	16	14	4	138	85	8	24	12	11	11

TABLE II — *Diabetic Group — Diabetic Retinitis Combined with Other Retinal Pathology*

DIAGNOSIS	NUMBER OF CASES TOTAL	NUMBER OF CASES SINGLE FUNDUS CONDITION	AVERAGE AGE YEARS	SEX MALE	SEX FEMALE	AVERAGE DURATION OF DIABETES YEARS	SEVERITY OF DIABETES MILD	SEVERITY OF DIABETES MODERATE	SEVERITY OF DIABETES SEVERE	AVERAGE BLOOD PRESSURE SYSTOLIC	AVERAGE BLOOD PRESSURE DIASTOLIC	CARDIO-VASCULAR RENAL PATHOLOGY PRESENT	CARDIO-VASCULAR RENAL PATHOLOGY ABSENT	COOPERATION OF PATIENT IN TREATMENT GOOD	COOPERATION OF PATIENT IN TREATMENT FAIR	COOPERATION OF PATIENT IN TREATMENT POOR
1. Arteriosclerotic	8	—	55	3	5	12	1	7	0	185	101	6	2	3	2	3
2. Albuminuric	3	—	57	1	2	13	1	2	0	150	86	1	2	1	0	2
3. Sclerosis of Retinal Vessels	1	—	49	0	1	4	0	1	0	146	104	1	0	0	0	1

TABLE III — *Control Group of Non-Diabetics*

DIAGNOSIS	NUMBER OF CASES	PERCENTAGE	AVERAGE AGE
Arteriosclerotic Retinitis	6	4	54
Albuminuric Retinitis	3	2	52
Moderate Sclerosis of Retinal Vessels	37	25	49.5
Advanced Sclerosis of Retinal Vessels	3	2	50.5
Negative	101	67	49

COMMENT

A number of interesting correlations may be made from our data:

1. The incidence of retinitis, and especially of the diabetic type is much higher than is generally reported in the literature. Statistics vary from 4.8% by Camidge[10] to 23% by Shepardson and Crawford[11] and 38% by Gresser.[12] We believe that the greater frequency of retinitis in our series is in part due to the fact that all our cases were minutely studied by the same individual. Wide dilation of the pupil and bright illumination is necessary, lest some of the punctate hemorrhages or small exudates in the macula be overlooked. Sometimes we missed them in one eye, but having found them in the other eye, we repeated the examination of the first eye and detected there, also, a few small exudates, or punctate hemorrhages, or both. Repeated examinations are also important in this connection. About ⅗ of our cases (89) were examined more than once, and 13 of these three times, during the course of our study.

2. Retinitis may occur in diabetes in the apparent absence of complications. This point has been the subject of great controversy with the preponderance of opinion of late leaning toward the negative side. While, on the one hand, Hirschberg,[1] Lagrange,[13] Friedenwald[14] and Gifford[15] believe that glycosuria alone is capable of producing retinitis; Foster Moore,[16] Cohen,[17] Wagner and Wilder,[18] Dirion,[19] Shepardson and Crawford,[11] and Camidge[10] claim that retinitis in diabetics is due to vascular changes. Our findings show that out of 15 cases of retinitis punctata centralis 9, or 60%, had no cardio-vascular-renal complications and 8 had a low blood pressure. Of the remaining 6 cases, one had

only an occasional albuminuria, the other angina pectoris, leaving only four with generalized arteriosclerosis. Definite nephritic manifestations were absent in the entire group. Furthermore, not a single case of central punctate retinitis was observed in the control group, although 27% of retinal sclerosis, some advanced, was recorded there. We, therefore, agree with Hirschberg[1] that retinitis punctata centralis is to be considered as pathognomonic of diabetes.

3. Our tables show that retinitis tended to occur more frequently in the more severe cases of diabetes. Thus, twenty of our twenty-seven cases of retinitis were judged to be moderate or severe diabetics and only seven were mild. In the negative group, however, the ratio of mild, moderate and severe was 16:14:4. The duration of diabetes and the age of the patient, on the other hand, had apparently very little relationship to the occurrence of retinitis, especially of the diabetic type. However, the group of cases with combined fundus lesions did show a somewhat longer average duration — twelve to thirteen years; and the average age was a little lower in the negative group (49) as compared with the retinitis group (55-57). The average blood pressure was higher in the retinitis group:

$$\frac{150}{90} \text{ to } \frac{170}{90}$$

than in the negative group:

$$\frac{138}{85}$$

This is somewhat at variance with the conclusions of Gresser[12] and Gray,[20] who thought that neither the severity nor the duration of diabetes were direct factors in the production of retinal pathology.

4. In regard to sclerosis of retinal vessels without retinitis, we found only a slightly higher incidence in the diabetic group as compared with the control group, the percentage being 34 for the former, and 27 for the latter. This is quite contrary to the findings of Pinas[8] and Gresser,[12] who found vascular sclerosis in 90 to 100%. However, the comparatively low percentage in our series is probably due to the exacting criteria we set for ourselves, requiring, as stated above, a combination of four to five signs before we called a case moderate sclerosis, while the above-

named authors considered cases to be sclerosis when only one or two signs were present.

In general, the sclerosis found in the entire group was of a very mild type. Marked arteriovenous constriction, silver-wire arteries, irregularity in vessel caliber, and perivasculitis were found only in rare instances, and then only in unquestionable cases of generalized arteriosclerosis.

5. The influence of adequate diabetic management upon the incidence and severity of retinitis may be judged from our figures on the degree of coöperation rendered us by the various patients. In the group of cases with retinitis the coöperation was largely poor and the ratio between the number of cases showing "good," "fair" and "poor" coöperation was 8:7:12. In the negative group this ratio was 12:11:11, about evenly divided. In several cases where the coöperation was good there was an improvement and, in two cases, even a disappearance of the retinitis. On the other hand, out of twenty-three cases which showed an aggravation of retinal pathology on subsequent examinations, only three had coöperated well. One of these was a case of diabetic-arteriosclerotic retinitis with a 220 blood pressure and 345 blood sugar in a diabetic of twelve years' duration. The other was a diabetic retinitis with a blood sugar of 205 and blood pressure of 160 in a diabetic of eight years' duration. In both these cases, in spite of good coöperation, the treatment, because of economic and social difficulties, could hardly be called adequate. The third case is difficult to explain since it was that of a diabetic of only two years' duration with a normal blood pressure. The other twenty cases, showing aggravation of retinal pathology, had coöperated poorly, or at most to a fair degree.

It is, therefore, apparent that proper diabetic management will lessen the incidence of retinitis or improve it when present.

6. It is interesting to note that about $\frac{2}{3}$ of the group with central punctate retinitis had never applied to our eye clinic for treatment and the remaining $\frac{1}{3}$ came in on account of refractive errors or beginning cataract. Most of the patients in the former category had a vision of 20/20 or better. These facts seem to indicate that central punctate retinitis is a very early manifestation of retinal pathology.

7. It is evident from these facts and those brought out in paragraph 5 that cases of diabetic retinitis offer a good prog-

nosis as to vision, provided they are properly managed and coöperate well. In advanced cases, where arteriosclerotic or albuminuric retinitis is added to the picture, treatment is of little avail. It behooves us, therefore, to urge the general practitioner and internist to have the eyes of their diabetic patients examined frequently. This should not only help the physician to gauge the adequacy of his treatment, but will probably, also, influence the patient to coöperate more carefully with his doctor.

Summary and Conclusions

1. A study of the fundi of 150 diabetics and a control group of 150 non-diabetics is reported.
2. The correlation of certain retinal conditions, with the age, duration, and severity of diabetes and diabetic complications is brought out.
3. The incidence of retinitis in diabetics is greater than is commonly believed.
4. Diabetic retinitis is a definite entity and occurs in diabetes in the apparent absence of its complications.
5. The incidence of sclerosis of the retinal vessels is but little higher in diabetics than in non-diabetics in the same age groups, and when present is usually less severe.
6. The prognosis of diabetic retinitis is comparatively good, provided the case is properly managed and coöperates well.
7. Effective diabetic therapy should lessen the incidence of retinitis.
8. Frequent fundus examinations are of particular value to the diabetic patient.

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DISCUSSION

Dr. S. Hanford McKee, Montreal: In the fundus examination of 1,272 diabetics at the Clinic for Diabetes at the Montreal General Hospital, of which Dr. I. M. Rabinowitch is the director, there were found 127 persons with retinal lesions, as follows:

Retinal Hæmorrhages	22
Retinal Arteriosclerosis	34
Neuro-retinitis	9
Retinitis	39
Diabetic Retinitis	21
Proliferating Retinitis	1
Lipæmia Retinalis	1

All sorts of senile and arteriosclerotic retinal lesions occur in diabetics as well as in non-diabetics. Nevertheless, it seems definitely established that there is one particular form of retinitis which occurs more frequently in those arteriosclerotics who have diabetes, than in those who have not. It would seem that there is some causal connection between the diabetes and the type and distribution of the arteriosclerotic process that accompanies it. In our series of 21 patients with diabetic retinitis all except 5 were also suffering from cardio-vascular renal disease.

Lipæmia occurs in a number of widely differing diseases such as: chronic alcoholism, phythisis, asphyxia, nephritis, phosphorus poisoning, pneumonia, peritonitis, gout, starvation and diabetes. Lipæmia of the retina, however, is a rare condition. It was seen once in this series of 1,272 cases, and is probably the only retinal lesion pathognomonic of diabetes. It occurs when the fat content of the blood rises above 4%. This condition occurs *chiefly* in diabetes, but, apparently, not necessarily *always*, as has been shown by Wagener's Report of Lipæmia of the retina in a case of Lymphatic Leukæmia, where the total blood fat reached 5%.

Evans believes that the first retinal lesion that occurs in diabetes consists in yellowish white spots near the macula, unaccompanied by hæmorrhages. They are in appearance, primary situation and method of spread the most characteristic lesion of diabetic retinitis. He states, also, that they are the earliest specific sign. This has not been my experience.

In a recent investigation by Rabinowitch, Ritchie and McKee at the Montreal General Hospital, the records of 1,500 diabetics taken at random were examined and tabulated. It was found that examination of the fundus alone was not sufficient to detect vascular changes in all cases, though it did detect the condition more readily than x-ray examination of the size of the heart with a six foot plate, or x-ray examination of the blood vessels of the lower extremities for calcification. Arteriosclerotic changes were disclosed by examination of the fundus in 37.1% of the cases; by calcification of the arteries in 30.5% and by x-ray of the heart in 18.1%. By a combination of all methods the incidence of arteriosclerosis was found to be 62.6%. This emphasizes the importance of using every available method for the detection of arterial disease.

The importance of this combination of methods is shown in a study of the relationship between duration of the diabetes, and the incidence of vascular disease. Of 500 cases in which all methods were used, 356 had had diabetes for less than 5 years, and of these 193, an incidence of 55% had vascular changes. To exclude the effects of age, the cases were again divided into those of 50 years and under and those of over 50 years. Of 162 cases of 50 years and under, vascular disease was detected in 64, an incidence of 39%.

One must not regard every arterial change noted in the diabetic as of diabetic origin; age and family history are important contributing factors. Doctors Folk and Soskin noted a greater incidence of retinitis in the poorly controlled diabetic. This coincides with our experiences published in 1929 with regard to the relationship between the degree of control of the diabetes and cholesterol and lends support to the view which is rapidly gaining ground, namely, that increased blood cholesterol is an important etiological factor in the production of arteriosclerosis.

Before the discovery of insulin, the duration of life from the onset of diabetes was but a few years. With better control by diet and insulin, the diabetic now lives long enough to develop cardio-vascular disease, whereas, in the past, death from coma or tuberculosis prevented him from so doing (Rabinowitch). In view of this change, one can readily appreciate the importance of detecting vascular disease early.

DR. A. D. RUEDEMANN, Cleveland: This excellent clinical study of the ocular fundi in diabetes made by Doctors Folk and Soskin will aid materially in bringing a proper interpretation to the pathology of the eye in this disease. Studies such as this are essential and constitute the proper method of investigating a disease whose disturbances are so general.

The situation with regard to diabetes is constantly changing and ever-varying reports must be expected. The disease is being diagnosed earlier, more frequently, and the chemistry of the blood is being worked out in so much greater detail that ophthalmologists must study the problem with a diabetes specialist in order to get the complete story of the physical changes.

Over a period of ten years we have subjected diabetic patients routinely to fundus examination, and have noted the changes in well-established cases of diabetes and in new cases in which diabetes was discovered in the course of examination for some other condition. I might say here, parenthetically, I didn't find very many of them myself. The widespread use of insulin and well-regulated diets has altered the picture and when these patients are seen early in the course of the diabetes, there are no significant changes in the fundi.

Diabetes produces changes in metabolism and blood chemistry, and the effects of these chemical changes may be seen in an advanced case of diabetes along the walls of the blood vessels, in the adjacent tissue and sometimes in the lens and other parts of the eye. There are a number of variations with which we must acquaint ourselves, such as diabetes and hyperthyroidism, diabetes and nephritis, senile arteriosclerosis and diabetes and the transitory sugar upsets occurring in shock and other severe constitutional disturbances.

Much of our knowledge of fundus changes depends on the ability of the examiner and the completeness of his examination. For the past three years we have added the study of the conjunctival capillaries to the study of the fundus in all types of cases and we have noted several interesting things. In the group with punctate macular hemorrhages, the hemorrhages also occur in the conjunctival capillaries and are of the diffusion (diapedesis) type. The capillary hemorrhages are frequently associated with central macular hemorrhage (as they are in senile central sclerosis) and may change the prognosis of a patient with cataract and diabetes. Again, I might state that in this group of cases, whether they are well regulated or not, even though the blood sugar is held to a very low level, I have seen a number of these patients go on almost to total blindness. The vascular degeneration is already so great that it is progressive, and the patient does not recover a great deal of vision. These patients are by far the poorest ocular risks, as diet and treatment do not completely control the recurring hemorrhages. There is a progressive vascular degeneration unlike that seen in other types of diabetes.

In another group with lipemic retinitis, the fat in the retinal vessels may disappear while the finer vessels still are covered with a definite yellow band for some time after the blood fat is normal. Which, I believe, demonstrates the fact that although the blood fat, according to our present methods of estimation, is normal, the body has not rid itself of the fat in the tissues.

Diabetes affects the blood carrying system and naturally causes definite vascular changes, even though in some cases senile sclerosis may produce diabetes, due to pancreatic island sclerosis or anemia; generally, the vascular changes in diabetes are secondary to the disease, to its duration, to the severity of its toxic process and to the general body reaction. The vascular changes are of diabetic origin and should be a part of the diagnosis. The vessels of diabetic children have not suffered the insults of repeated infection or faulty metabolism and naturally are still in good condition although, even in the young, the capillaries are not entirely

normal. Further studies of the capillaries in diabetes will reveal more pathologic changes and will add to our knowledge of the changes in the retinal blood vessels.

DR. M. L. FOLK, closing: I wish to thank Doctors McKee and Ruedemann for their kind discussion. I haven't very much to add, as they, practically, agree with me. Dr. McKee gave his findings as 37% of sclerosis of the retinal vessels in the group, and we found 34%, which is a pretty close finding.

As for hemorrhage, mentioned by Dr. Ruedemann, I might say, in our series, we have found very few extensive hemorrhages in the retina in the cases called diabetic retinitis. We found them in some of the combined cases of diabetic-arteriosclerotic or diabetic-albuminuric retinitis. We have not made capillary studies, but, I might say, the blood-chemistry was checked very frequently, and the personal interview, by Dr. Soskin of his patients, was done as often as possible, so, I think, we have had a pretty good estimate of the diabetic status in these cases.

SOCIAL SERVICE IN OPHTHALMOLOGY

ELEANOR P. BROWN*
(By Invitation)
NEW YORK

AND

LAWRENCE T. POST, M.D.
SAINT LOUIS

EIGHT YEARS AGO one of us had occasion to give a report on the treatment of glaucoma in the Eye Clinic of Washington University. A review of the cases seen in the clinic for the preceding ten years brought to light some amazing facts. Less than 50% of these patients had made more than one visit to the clinic and less than 5% were followed for more than six months. In those days there was no social service worker in the clinic. Today there are two workers and the story is quite different, for it is most unusual for any glaucoma patient to drop from sight in the first six months of treatment.

The general average of patients coming to operation (when such has been recommended) is said to be about 30%; records in the Washington University Eye Clinic show that 95% of the patients for whom operation is advised actually have the operation performed!

We would not take the time of this busy organization to present this paper did we not feel so strongly that the social worker is a necessary adjunct to an efficiently conducted clinic and did we not believe that ophthalmologists, who have in general been so keenly progressive, have been unthinkably slow to make use of her service.

You may ask in what ways can the trained social service aid be of assistance to the doctor and why is the type of training that she has had the most desirable for this coöperation with the ophthalmologist? To answer the second question first, an essential to the success of any treatment is the patient's coöpera-

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tion. This may be totally lacking, insufficient or at least far short of maximum, because of failure to understand the medical abnormality or the intent of the treatment; because of inability to adjust home conditions to the needs of the case, or because of general lack of comprehension of the entire situation. In bringing about this desired coöperation, the social worker is an expert. Her training has been based on the study of human reaction to given situations, with a thorough appreciation of the varied response from different individuals.

And now let us consider what a social worker can do for the ophthalmologist: In the clinic program a social examination made of new patients is important and will prevent many of the failures to follow up medical directions. Not only is the advice given in this preliminary interview helpful, but from the very start the patient feels that he has a friend and intermediary who will be of assistance as long as he must continue the clinic contact and who will be an ever-present help in time of trouble. In Washington University Eye Clinic the social workers in 1933 interviewed 60% of approximately 5,000 new patients.

Instructing the patient so that he may get most benefit from the clinic, though the simplest type of social help, is constantly needed and brings real satisfaction from the standpoint of end results. Occasionally there is the necessity of modifying the patient's behavior or that of his family. To do this efficiently requires technical training.

With understanding of the doctor's findings the social worker can add emphasis to the recommendations that have been made and explain their significance in relation to the particular eye condition. At the same time she can acquaint the doctor with his patient's social and economic status so that any disturbing material or emotional factors contributing to the physical state may be duly considered. In the adjustment of such social and economic problems as may exist, the social worker, of course, has prime responsibility. It is easy to see how important a part such adjustments can play in the treatment of certain eye patients — from those in whom constant fear and worry are creating a state of mind that brings harmful pathological reaction, to those who for financial reasons cannot obtain the advantageous diet, drugs or lenses that are prescribed. The large number of patients scheduled for continued treatments but lost sight of in the pressure of clinic work is a burden on the mind of any

conscientious doctor; under a follow-up system planned and put through by a social service department, such cases can be followed and the danger of neglected treatment be avoided. We have all experienced the disappointment of refused operations, the failure of patients to accept the hospital care advised; here, too, the social worker can help materially in paving the way to complete medical and surgical care.

In research problems, a social worker with an understanding of eye conditions is prepared to assist the ophthalmologist in collecting and compiling material gathered from hospital and clinic records. Given the requisite background, she can help in analyzing case material.

There are numerous specific diseases in which the social worker can be of especial value, of which glaucoma is typical. A recent five-year report of intensive work with glaucoma patients at the Massachusetts Eye and Ear Infirmary, where a social worker was put in charge of this one group of patients alone, divides her work into three distinct phases: (1) the gathering of data for study; (2) the following of all patients to insure uninterrupted treatment; (3) the carrying through of such social treatment as is needed for successful medical care. During three years, average daily attendance grew from seven to thirteen, and the lapsed cases on record, together with those under more or less regular surveillance, were systematically traced and brought under control. This same report shows steady increase in the number of primary glaucoma cases admitted for operation — 55 in 1928, 115 in 1929, 208 in 1932. The significance of this increase is evident to any ophthalmic surgeon who knows the tendency of clinic patients to avoid operative care and the consequent hopelessness of sight conservation.

Syphilis is a tremendous factor in the incidence of lost and impaired vision. Syphilologists and ophthalmologists alike have more responsibility for its eradication than simply the provision of medical treatment. Treatment without education as to the dangers of the disease to patient, family and to the public at large, is but a half-way measure. The social service worker's skill in interpreting medical findings and recommended procedure, in bringing about the proper tie-up between eye and syphilis departments, in ensuring continuance of treatment over a long period in the face of various and sundry difficulties is a much-needed element in the control of ocular syphilis. As a step toward the prevention of

congenital or prenatal syphilis so frequently resulting in damaged eyes, the American Medical Association on June 15, 1933, approved the appointment of a committee to take the matter under advisement with a resolution as adopted by various sections of the American Medical Association calling for the development of such coöperative medical, social and public health measures as may eventually bring about the treatment and care of all infected pregnant women. Just within the past month this educational campaign has started, so ophthalmologists should be alive to the situation and should make every effort to help in this great preventive undertaking.

With tuberculosis of the eyes and with trachoma, the social and public health aspects are also self-evident as is, again, the need of educating the patient and the family group. Interpretation and the winning of confidence in medical procedure may make possible the operation required in a cataract case, senile or congenital. The doctor has neither time nor opportunity to contribute this service or to put through the plan for improved hygiene, economic security or educational adjustment that may be called for. Children with progressive myopia, for whom a program of specialized education based on the proper safeguarding of vision is so all-important, must be followed.

In spite of existing laws or regulations in practically all of our states providing for the use of a prophylactic in babies' eyes at birth, children are still entering schools for the blind, sightless because of ophthalmia neonatorum. A recent etiological and topographical classification of causes of blindness in 17 schools for the blind in the United States, issued by the Committee on Statistics of the Blind, gives this as the predominant item under infectious diseases, with prenatal syphilis running a close second. What an opportunity ophthalmologists have, coöperating with social workers, to reduce this needless waste!

The same table shows traumatic and chemical injuries as responsible for 7.8% of the school entrants — a fact indicating the part that a well-prepared hospital social worker can take in building up a community consciousness of eye problems and their alleviation. Someone close to the eye physician in his hospital and clinic practice, seeing the types of cases passing through the department and understanding their significance in relation to public health and the social, industrial and educational life of the city or locality from which they come, can do

much toward bringing about desired improvements where needed — such as adequate provisions for the eye health of school children, eye protection in industry and prenatal care.

Though medical social work with eye patients began in this country in 1907 with the organization of a social service department at the Massachusetts Charitable Eye and Ear Infirmary, ophthalmologists have been slow to realize its significance, and its growth has been small in proportion to similar service in other fields of medicine. In 1922, Dr. Conrad Berens brought out the need for fuller recognition of social service in a study made by the Section on Ophthalmology of the Associated Out-Patient Clinics of New York City. Here agreement was expressed with the statement of Dr. George S. Derby that "no hospital at the present day which serves the people can give really efficient service unless it makes provision for social service work and establishes some sort of follow-up system." Through the years certain other progressive ophthalmologists have emphasized the value of social work — notably Dr. George de Schweinitz, who, in 1915, called public attention to its vital place in clinic practice. But with unbelievable slowness have eye hospitals and out-patient departments accepted the service as a need, and this in spite of evidence through the New York report and through showings elsewhere that a high percentage of eye patients suffer unnecessarily from visual loss because of neglected treatment and failure to receive social care.

Belief in hospital social work as a factor in preventing blindness was the urge that brought about employment of that first eye worker in Boston in 1907, and it was with the same belief that years later, in 1931, the National Society for the Prevention of Blindness undertook intensively to stimulate the more general employment of qualified personnel in this field. From Boston again came the initial step, when Dr. Derby, of the National Society's directorate and Professor of Ophthalmology at Harvard University, offered the facilities of the Massachusetts Eye and Ear Infirmary as a training center for selected medical social workers to specialize in eye conditions. The National Society responded to the idea of Dr. Derby by granting scholarships to the students selected and by providing staff service in administering the project.

It is our firm belief that a medical social worker serving in the field of ophthalmology should have special preparation in

order to function effectively. Without some understanding of the eye and its involvements, how can she interpret the doctor's findings; how can she intelligently plan a program of adjustment for the patient without a realization of the conflicts growing out of a visual handicap and a knowledge of resources that may be used or developed for the patient's welfare? Furthermore, if she is to be used, as she should be, for creating a community interest in sight conservation, she must know whereof she speaks. However, we do not mean that only in special courses of training can the required knowledge be obtained. A high-grade medical social worker can, through actual experience in a well-organized eye department gain a thorough understanding of her work and develop expert technic; but the process is a long and, to our minds, a wasteful one. In the interests of economy and of first-class service, intensive training courses have been sponsored by the National Society for the Prevention of Blindness and will be available this fall to a limited number of qualified candidates who have positions in view. To date, 13 medical social eye workers have been prepared in Boston and at a second four-months' training course established later at Washington University, St. Louis; perhaps the number would have been larger but that it is far from the intention of those who are responsible to provide trained workers for whom there is no demand, and ophthalmologists calling for social workers are few and far between.

In conclusion, we most earnestly urge ophthalmologists to awaken to the tremendous advantage to be gained through the aid of social service, and to avail themselves of any opportunity to secure this most valuable coöperation.

DISCUSSION

Prof. Dr. Hans Lauber, Warsaw, Poland: It is exceedingly interesting to hear what social help can do. In Europe we also realize this problem, but we are under great difficulty in executing the program. Some cities already have in their hospitals social workers who help to follow up cases, control them, bring them in, according to the line which has been explained by Miss Brown.

One can approach prevention of sickness from another point of view. In the city of Vienna, the municipality has decided that every man or woman who is employed in the service of the municipality has to undergo a thorough medical examination before appointment. In every case there

is a Wassermann test made. This has proved to be very beneficial, because, by this means, especially by the making of the Wassermann test, one has been able to detect quite a number of individuals whose health was not sound, and whether or not they were taken into the service of the municipality, they were followed up and were prevailed upon to submit to efficacious treatment.

Hospitals can provide other ways to help in the prevention of disease. We have made it a rule — and I know that some other clinics do it; they do it in Vienna; I do it at my clinic in Warsaw — that every patient (unfortunately, this only applies to the indoor patients not to the outdoor patients, because we haven't the means nor the personnel to put that through) has a Wassermann and a tuberculin test made. One is reviewed from the internal point of view, quite independent of what his affection may be. So we detect, of course, in cases of cataract, cases of leukemia, ulcers, and detect some others, let's say, lues concomitant, or strabismus which has latent lues, or something of the kind.

By these means one can also help partly to supply the social worker, which, unfortunately, I have not got at my clinic, and I think it is practically outside the scope of hope that I shall ever be able to secure anything of that kind excepting, perhaps, as they control carcinoma by the voluntary aid of the ladies of the society.

Section on Otolaryngology

The American Academy
of Ophthalmology and Otolaryngology

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REPORT ON LABYRINTH WORK*

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MONTREAL, CANADA

BEFORE BEGINNING this report, I should like to make it clear that the experiments about to be described are the result of a conjoint experimentation carried out by Dr. Tate, Dr. Ross and myself, all of the Physiology Department of McGill University. I would like to make it quite clear, that these are not only my experiments, but theirs also, and that it is with their consent and their assistance that I have prepared, for your information, this rather brief outline of our work.

We have been, for a long while, occupying ourselves with the question of turntable rotation, or what is usually thought to be the question of horizontal canals. For a long time we have realized that the question of turntable rotation is rather complex and that it does include more than horizontal canals. During the past year we have approached the problem from three different angles.

The first approach was an attempt to record the head movements and deviations that result from turntable rotation. The second approach was to observe and photograph (we used a moving picture apparatus) the reflexes that result from turntable rotation after having severed the nerves to the individual receptors in the labyrinth.

The third approach was one which was a little more involved than the others, the question of the study of action potentials from the individual receptors of the labyrinth.

In the first part of the experiment, we devised an instrument whereby, when the animal was subjected to ordinary turntable rotation, it was possible to record the movements of its head.

With this simple apparatus, the frog is placed on the table, the little stand on which the hairpin is attached. The tip of the frog's nose is cocainized. A hook is placed in the nose and,

* Research grant by Council.

from the hook, a string leads to the top lever. The bottom lever records the movements of the table. In that way it is possible to have a graphic representation of any movement that the frog's head may make in response to the rotation.

The interesting thing that came out of this research was that, in addition to there being a head deviation when the horizontal canals were present, there was also movement of the head when the horizontal canals had been removed, that is, by sectioning the nerves to each canal. We found, upon analysis, that that movement followed even after all the labyrinthine receptors were removed and only the two posterior canals remained. If both labyrinths were removed, there was no longer any head deviations. So it became quite obvious that, in addition to the horizontal canals, the vertical canals also caused a head movement, and what is more important is that the vertical canals are apparently being stimulated by a movement at right angles to the plane in which they lie and to the plane in which they are ordinarily considered to be stimulated.

In the second part of the experiment, as I say, we used moving pictures in order to be able to observe and study the reactions even after the experiment had been finished. In that series of experiments we tried to study each individual part of the labyrinth separately; that is, for instance, first of all we removed both horizontal canals in an otherwise intact animal. When that animal was turned on the turntable, he gave no response whatsoever to the turning.

In another animal we removed all the labyrinthine receptors except the two horizontal canals. The interesting thing about this frog is that he responds quite well to the turntable rotation, but it is a most abnormal response. His head is no longer steady; it is wobbly. His whole movements are very jerky and most abnormal; so that, evidently, there must be some mechanism whereby the head is steadied during the movement.

If all the semicircular canals are removed, leaving only the utricle intact, and the frog is subjected to the turntable rotation, if it is over the center or the axis of rotation, there is no reflex reaction. One is unable to see the frog do anything at all, so that one is forced to conclude that to ordinary turntable rotation, or spin, the utricles are not responsive.

On the other hand, if one removes the utricles and leaves the canals intact, immediately on moving the table, on turntable

rotation, there is a definite reaction, but now, instead of there being the ordinary lateral deviation, there is, in addition, quite a torquing or bending effect of the whole body. So that, evidently, some other factor is coming in now, in addition to the horizontal canals.

If we section the horizontal canals and have nothing left but the vertical canals, again we see the vertical canals being stimulated by rotation, turntable rotation, or rotation by the vertical axis.

In order to localize this still more, we left, in turn, an anterior vertical canal in one case and a posterior vertical canal in another, no other labyrinthine receptors being intact. In each case the vertical canal showed a very definite response to the rotation about a vertical axis. Incidentally, the posterior canal gave a better response than the anterior.

In order to check up on this work, we did the action potentials. Just in order to refresh your minds, I would like to point out that when any nerve is carrying an impulse, at the moment that the impulse passes along the nerve, there is a change in the electrical potential of the nerve.

If the wire from a galvanometer is placed on the nerve, at the moment that the impulse passes the wire, of course the current will be picked up by this wire, and it is possible to conduct it to a galvanometer and actually photograph the electrical change.

Naturally, in dealing with an end-organ as small as the labyrinth, and if one is going to attempt to record the action potentials from the individual receptors of the labyrinth, the technical difficulties become very considerable. However, we were fortunate enough to be able to photograph and to hear the action potentials from each individual receptor in the frog's labyrinth.

The mechanism we used was the usual type of Weaver and Bray apparatus, but in this instance we were not so much interested in the sound as we were in the vestibular part of the wire. The active electrode, the platinum electrode, was attached to the cut end of the frog's eighth nerve. The wires were then led to an amplifier which amplified the electrical current, the voltage of it, about 200,000 times. This was then led to an oscillograph, and the impulses or the deflections of the oscillograph were photographed.

In addition to this, we had connected up in the circuit a loudspeaker so that we were able to hear the electrical potential changes as they occurred in the nerve.

The experiments themselves may be divided into two sections. That is, we examined, first of all, the otoliths, and, secondly, the semicircular canals.

In order to examine the otoliths, we cut the nerves to all the canals, and if we were examining the utricle, we then cut the nerve to the saccule and removed the saccule, so the only nerve intact was the nerve to the utricle.

This was then connected up to the apparatus and placed upon a tilt-table. Then if it was, for instance, a utricle, we found that tilting the preparation gave the maximum response. However, as I told you just a few moments ago, we found that with a frog on a turntable, if all his canals were removed and only his utricles were intact, simple rotational spin gave no visible result. However, when we had the preparation attached for action potentials, we did hear and could photograph some impulses. That, therefore, suggests that the utricle is doing something, but evidently not sufficient to bring about a true reflex.

In passing, I might just say that when we had the saccule connected up for action potentials, the only test to which the saccule really responded was vibration responses to forks from about 128 to about 512. We could not be absolutely certain that we got any responses from the saccule from tilting or rotational tests.

When the individual canals were connected up in the circuit, they gave, of course, the best responses to tilting in their own plane.

In a photograph of the action potential changes from the posterior vertical canal to tilting backwards in the plane of the canal, we see three little irregular deflections, which are three different tilts of a posterior canal.

In addition, however, to the response of the canal in its tilting in its own plane, it gave a very definite response to rotation about a vertical axis, that is, ordinary turntable rotation. Actually, the threshold of rotation was higher for a posterior vertical canal than it was for a horizontal, but the response is very definite.

In addition, there was a very definite response from the anterior vertical canal. So that, from the ordinary head deviation recordings, from the observation of the reflexes, and from the action potentials, we get a definite response from the vertical canals to rotation about a vertical axis or ordinary turntable rotation.

If I may summarize, then, it would seem that, in order that the vertical canals and horizontal canals may function normally, particularly the horizontal canals, the head must be kept level by the utricles and by the vertical canals, and then the horizontal canals come in and prevent any counter-circling or head deviations in the case of spontaneous movement, or, if it is in response to rotation, the horizontal canals actually elicit these counter-reactions, the effort apparently being for the animal to be able to retain its original field of vision.

These experiments are confirmed by the recorded head deviations, by the photographing of the ordinary reflexes, and by a study of the action potentials.

In closing, gentlemen, I would like to take this opportunity of thanking the Academy for the very real assistance which they have given us in the carrying out of this research.

AGRANULOCYTIC ANGINA FOLLOWING INGESTION OF AMIDOPYRINE OR THE BARBITURATES

CHARLES H. WATKINS, M.D.*
(By Invitation)

ROCHESTER, MINN.

PRIOR TO the report by Madison and Squier in 1933 of thirteen consecutive cases in which amidopyrine, or some of the barbiturates combined with amidopyrine, apparently were etiologic factors in the production of primary granulocytopenia, the etiology of this condition had been a matter of a good deal of controversy and speculation, although Kracke in 1932 suggested the possible etiologic rôle of preparations containing the benzene ring. It was generally believed that the disease was the result of failure of the myeloid elements due, on the one hand, to an overwhelming infection, or toxemia, or, on the other, to primary disease of the bone marrow.

Following this report, I, in 1933, reported a series of cases in which there seemed to be a possible relationship between the ingestion of drugs containing amidopyrine and of certain of the derivatives of barbituric acid, and the onset of granulocytopenia. In this report it was stated that in most of the cases of agranulocytosis the patients had either complained of fatigue or had had some chronic disease for which they had been receiving medical care. Since the time of that report, numerous reports have been made indicating that amidopyrine is an important factor in the production of granulocytopenia. Rawls; Holten, Nielsen, and Transbol; Randall; and Hoffman, Butt, and Hickey have reported a total of twenty-one cases of the disease in which amidopyrine itself or amidopyrine in combination with other drugs had a real relationship to the development of the disease.

Zinninger recently reported two cases in which the patients had been taking large amounts of a compound containing amytal

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for long periods of time. More recently, Reznikoff has reviewed the literature and has discussed the sensitivity to amidopyrine of patients who have agranulocytosis. Fitz-Hugh has discussed the possible etiologic rôle of amidopyrine in the production of granulocytopenia, and he concluded that compounds containing amidopyrine may have been contributing causes in more than half of his series of cases of agranulocytic angina. Of his twenty-six patients, seventeen were known to have ingested amidopyrine just before the onset or during the course of the disease. He stated, however, that since ingestion of amidopyrine had not always produced agranulocytic angina, it of itself could not be accepted as the sole cause of the condition. He regarded reaction to this drug as an allergic phenomenon, so far as leukocytic behavior is concerned, and this is particularly borne out in the results obtained in experiments on animals. It is certainly true that, in some cases, agranulocytic angina is not due to ingestion of any of these preparations, nor to any other medical agent. Heck has reported a case in which there were repeated attacks of the disease. The patient in this case has been under his observation for about ten months and at no time has taken any drugs that might in any way be a causative factor in the condition. The fact that the disease is so rare and that so few individuals seem to show the agranulocytic phase following ingestion of these substances, suggests an idiosyncrasy rather than a general reaction to such preparations.

I wish to report a series of fifty-three patients with true agranulocytosis who have been observed at The Mayo Clinic. Of these fifty-three patients, eighteen had taken amidopyrine, either alone or in combination with other sedative drugs, but exclusive of the barbiturates. Two of these eighteen died, having taken preparations containing amidopyrine during the entire course of their illness.

The blood count for all patients in this series had been normal prior to administration of the drug, although most of these patients had been chronic invalids for long periods of time. No patient in this series who recovered from an attack that had presumably been induced by amidopyrine has had a recurrence following discontinuance of the use of the drug.

One case is of interest in that the patient had twelve attacks of the disease and has recovered from each; she has been under observation for more than four years. This patient was

subject to migraine for which she had been taking amidopyrine. The attacks of agranulocytosis had come regularly and had been very severe; in all of them there was extensive ulceration of the mucous membranes of the mouth and pharynx. The leukocyte count in each instance was less than 1,000 per cubic millimeter of blood, with a total absence of neutrophils. Administration of all sedative preparations was discontinued in June, 1932, and no further attacks occurred until February, 1933. The only medication before this last attack consisted of 6 grains (0.4 gm.) of pentobarbital sodium (nembutal) which had been taken in two doses of 3 grains each (0.2 gm.) on successive days. Twenty-four hours after the second dose the patient had a chill, the leukocyte count dropped to 600 per cubic millimeter, there was total absence of neutrophils, and ulceration of the mouth developed. Recovery from this attack was very much slower than from those which had been induced by amidopyrine. The patient, however, has had no further attacks although she still has migraine; the blood has remained perfectly normal in every respect since use of these drugs has been discontinued.

In addition to the eighteen patients who have been mentioned, eight patients took amytal, or sodium amytal, prior to the onset of agranulocytosis; five of these eight died. One case in this group is of interest in that the patient recovered from two attacks, the first of which was probably induced by amidopyrine; the administration of this drug was discontinued because of its suspected etiologic rôle in the attacks. Because of insomnia, however, the patient was given amytal. Within forty-eight hours the blood count decreased and typical agranulocytosis developed; the patient recovered under treatment, although the response was much more prolonged than in previous attacks. Four months later, of her own accord, the patient took 6 grains (0.4 gm.) of sodium amytal for insomnia; a third agranulocytic phase developed from which she failed to respond to any form of treatment, although she did not die until twelve days after the initial drop in the leukocyte count. This patient failed to respond to treatment that had previously proved efficacious in her case. One other patient in this group recovered from two attacks induced by amytal, the first being very severe. Four months later this patient, of his own accord, in order to see if the drug would produce the condition, took 1½ grains (0.1 gm.) of amytal. Within thirty-six hours the leukocyte count had dropped to 1,800 per

cubic millimeter and the neutrophils to 1% ; later, following treatment, the blood rapidly returned to normal. The five patients in this group who died had taken sodium amytal; the three who recovered had taken amytal. From this observation it would appear that if administration of these compounds had any significant effect on the condition of the patients, sodium amytal carried a higher mortality risk than amytal.

Seven patients received pentobarbital sodium prior to the onset of granulocytopenia; four of them died. All of these seven patients had undergone either surgical procedures or hospitalization; blood counts of all seven had been normal prior to ingestion of the drug, and none of the seven had received any other medication.

There were four cases in which granulocytopenia developed after the patients had taken allonal*; three of these patients died. All four were patients who were constantly complaining, and they had been taking the drug for periods of at least two weeks prior to the onset of the disease; one of them had used allonal constantly for three months before the agranulocytic syndrome developed. The patient who recovered was being treated for angina pectoris and, because of insomnia, had been given allonal on three successive nights prior to the onset of the leukopenic state. His recovery was quite rapid after administration of the drug had been discontinued, and treatment for agranulocytosis had been instituted.

Two patients had received no other drug than phenobarbital (introduced under the proprietary name luminal). One of these two died in an attack; the other recovered. The patient who recovered had toxic dermatitis coincident with the leukopenia, the total leukocyte count being 350 per 100 cc. blood; neutrophils were totally absent. The dermatologist felt that the dermatitis was the typical result of an idiosyncrasy to phenobarbital, and as the patient had taken no other drugs, we assumed that phenobarbital, in this instance, may have produced the leukopenic state. Certainly if this idiosyncrasy to phenobarbital does exist, it must be exceedingly slight, as directors of several epileptic colonies have never to their knowledge encountered agranulocytosis among patients in their respective institutions, even among those who have used the drug for years.

* Allonal is a proprietary mixture of allylisopropylbarbituric acid and amidopyrine.

Typical agranulocytosis developed in one case following the patient's use of acetanilid. This patient recovered rapidly and has had no further attacks since abstaining from all sedative preparations.

Six patients had taken sedative drugs for headache prior to the onset of leukopenia, but the nature of such drugs was not known. Two of these patients died.

In six cases there either was no history recorded of the patients ever having taken any drug prior to an attack or else the fact whether they had or not could not be determined from questioning them; there were no fatalities in this group. The first patient in this group had had repeated attacks of quinsy and had been under the care of a physician; there was no note on the history, however, as to what type of medication had been given. The second patient had received 3 grains (0.2 gm.) of pentobarbital sodium during the attack, but there was no history of the earlier use of any drug prior to the onset of granulocytopenia. The third patient had been fatigued for six months prior to the onset of the leukopenia, but had not required any sedative preparations during this time or taken any other drugs which could be incriminated. The fourth patient apparently had received no medication, even though under a great deal of nervous strain. The fifth patient had had Vincent's infection, with marked hyperplasia of the mucous membrane of the gums; there was no note on the record in regard to any medication having been given. The sixth patient has been under observation for ten months, having had repeated attacks, none of which was induced by drugs.

Bohn has reported a case of agranulocytic angina following ingestion of dinitrophenol. In our series, one patient had received dinitrophenol prior to the onset of the leukopenia; she made an uneventful recovery following discontinuance of use of this drug.

In our series there was a high incidence of the disease among physicians, dentists, and nurses, and members of their immediate families. Many other investigators have noted a similar high incidence among professional people; this probably is due to the fact that they have more ready access to the drugs than laymen.

There seems to be strongly presumptive evidence in support of the theory that an idiosyncrasy to amidopyrine may pro-

duce agranulocytic angina. I also feel that the barbiturates, although the idiosyncrasy to these drugs probably is not so great, may, likewise, under certain conditions, possibly only among certain debilitated patients, produce this condition. Whether these persons have a primary deficiency of the bone marrow is under question, but in most instances, prior to the onset of the leukopenic state, the leukocytic response to infection and the general blood picture have been normal. The fact that there is never an anemia of any degree of severity in true agranulocytosis is supporting evidence for this contention.

The fact that in certain cases repeated attacks of neutropenia resulted following ingestion of these drugs demonstrates that there is an apparent association between their use and the neutropenic state. This possible relationship is not, in my opinion, however, a contraindication to their use. When they are used regularly, it would seem advisable to make a leukocyte count every two or three days while the patient is in hospital, and every two or three weeks after the patient's dismissal from the hospital if use of the drug is to be continued. The patient should be warned to look for early symptoms of the disease, consisting of fatigue, headache, and difficulty in swallowing. The latter results primarily from soreness along the sternocleidomastoid muscle, which generally occurs before there is any demonstrable irritation of the mucous membranes of the mouth or throat, and any chill or rapid rise in temperature. Should any of these symptoms occur, a leukocyte count and a differential estimation should be made. As has been stated previously, if agranulocytosis in many instances is due to any idiosyncrasy to these various drugs, it must be exceedingly rare, for the very great majority of individuals use these drugs without untoward reaction. A great deal of further clinical and experimental work will be needed to establish this thesis.

That these various drugs possess any etiologic significance in the production of agranulocytic angina, therefore, is still open to question. Kracke and Parker have maintained that drugs containing the benzene ring may be responsible for producing the disease, especially those belonging to the benzamine group. Since this group is not contained in the barbiturates, and as there appears to be a strong probability from this study that derivatives of barbituric acid may exert a similar influence on the myeloid leukocytes, the actual causative factor is still unknown.

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DISCUSSION

DR. JOHN J. SHEA, Memphis: Dr. Watkins has given us a wonderful presentation of a subject that to us as laryngologists is important. Besides the action of the barbiturates and amidopyrine on the hematopoietic apparatus, we as otolaryngologists must bear in mind that there is a true entity which is called Schultz' disease that is an agranulocytosis that, as far as we know, is an entity and is not caused by a drug.

It has been my misfortune to see three of these cases, all of which have died.

The first of these patients had only received quinine as any medication at all for the past year. The other two patients had received no drug whatever prior to the onset of the acute throat. The second patient I saw in consultation before you could make out a throat lesion sufficiently to justify his blood picture. His blood picture was dropping more rapidly than what the throat lesion would indicate. The third case was a typical one of Schultz' disease. She came into the hospital with the lesion on the anterior pillar of the tonsil that has been so thoroughly described in many of the articles, and she went on to death.

Of the other drugs that may present this depression of the hematopoietic system, arsenic has entered into two of these cases. Both of these patients received two doses of salvarsan and, on the second dose of salvarsan, developed the blood picture as shown here. These cases are not the Schultz' type. They are the types like the amidopyrine and the barbiturates.

TRUE SCHULTZ' TYPE

	Mrs. M.	*Mr. E.*	*Mrs. H.*
Drugs	Quinine	None	None
Lesions	Tonsil	Tonsil	Tonsil
W. B. C.	1200	3500	350
Polys.	10%	None	None
R. B. C.	3,240,000	3,670,000	3,580,000
Transfusions	Yes	Yes	Yes
Cacodylate	No	Yes	Yes
Pentnucleotide	No	Yes	Yes

SYMPTOM COMPLEX TYPE

	Mrs. P.	*Mr. M.*	*L. A.*	*Mrs. M.*
Drugs	Arsenic	None	Arsenic	Liver
Lesions	None	Tonsil	None	Nose
W. B. C.	4400	1800	6500	7000
Polys.	2%	1%	9%	8%
R. B. C.	3,300,000	3,700,000		3,310,000

The reason that the arsenic apparently has this depressing action on the hematopoietic system is the lack of sulphur in that individual. So that we as otolaryngologists must remember that there is a true agranulocytic angina that is not caused by drugs, and, on the other hand, there is this clinical complex that is caused by the barbiturates, amidopyrine, arsenics and other depressants of the hematopoietic system.

Dr. Gordon B. New, Rochester, Minn.: In the Journal of the American Medical Association under date of June 30, 1934, there is a report by the Council on Pharmacy and Chemistry, which states: "Apparently amidopyrine may cause granulocytopenia to occur in certain patients, but there is no definite evidence that barbital compounds alone are responsible."

Dr. Watkins' report is very important, as he has shown definite evidence that the derivatives of barbituric acid are responsible for granulocytopenia in certain cases, and I wish to compliment him on the excellent piece of clinical research. There is apparently strong evidence to incriminate the amidopyrine preparations as well, and I feel that it should be a routine procedure to have leukocyte counts made on all patients who are continuously taking these drugs, for in our experience an occasional case of agranulocytosis develops in this group and is recognized early only by the routine taking of blood counts. The prognosis in this type of case, if treatment is instituted before the ulceration appears, is much better.

I wish to stress three clinical diagnostic points in the early recognition of this disease, before ulceration of the mucous membrane is present. The earliest symptoms that we have been able to recognize are: (1) fatigue; (2) distress upon swallowing; and (3) pain along the sternomastoid muscles of the neck without sore throat. If a blood count is taken on these cases at this time it will reveal the leukopenia or the neutropenia. We all know that the disease is usually not recognized until there is ulceration in the pharynx or mouth. At that time marked toxemia is frequently present due to the secondary infection, and the prognosis from any form of treatment is not good. Dr. Watkins has brought out that if these cases are recognized early, before the ulceration and toxemia appear, he has been able to obtain improvement and some of them have remained well without return of the disease for one and one-half years.

In order to stress the fact that this disease is a systemic one that involves all the organs of the body, but in which the first symptoms may be referable to the throat, I am presenting a group of slides to show the ulceration lesions with pseudomembranous ulceration:

(1) Ulceration in the tonsil, pharynx and epiglottis, showing the pseudomembranous ulceration in these areas.

(2) Post-mortem specimen showing the ulceration at the base of the tongue and pharynx, as well as in the larynx.

(3) Pseudomembranous ulceration in the subglottic region and trachea.

(4) Pseudomembranous inflammation in the trachea and bronchi.

(5) Hemorrhagic edema of the lungs.

(6) Acute ulcers of the stomach.

(7) Acute ulceration of the ileocecal valve.

(8) Acute ulcers of the colon.

(9) Acute ulceration of the rectum.

In closing, I wish to urge early recognition of this disease before there is any ulceration of the mouth, pharynx or larynx by having blood counts made on patients who present symptoms of fatigue, distress in swallowing and soreness along the muscles of the neck; and that leukocyte counts be made on all patients who are taking amidopyrine preparations or derivatives of barbituric acid, so that those who have an idiosyncrasy may be recognized early and receive the value of efficient treatment.

DR. CHARLES H. WATKINS, closing: I wish to thank Dr. New and Dr. Shea for their discussion.

I would like to point out it is my feeling that the true agranulocytic disease of the Schultz' syndrome may also be produced by amidopyrine, as most of these cases I have seen presented all the cardinal features that have been described by Schultz. They have the ulceration, leukopenia, the high fever, and yet they do recover following treatment. We use a little different form of treatment than is used in general. We have used a preparation of bone-marrow, on which I reported before the A. M. A. about a year ago, with very satisfactory results.

I think that, without question, as Dr. Shea has also stressed, a good many of the cases of this condition do occur without any definite etiological factor that we know at the present time.

For this reason I wish to stress that we must have much more clinical and experimental evidence before we can be definitely sure as to the actual etiology of the condition.

SYMPOSIUM ON TONSILS

WHY AND WHEN THE TONSILS SHOULD COME OUT

ALBERT D. KAISER, M.D.
(By Invitation)

ROCHESTER, N. Y.

IF THE FUNCTION of the lymphoid tissue known as the tonsils were better understood or if there were some unanimity of opinion among those who have discussed the function of the tonsils there might be less justification for the pro and con discussion on when the tonsils should come out.

Pathologists and physiologists have described the diseased tonsil and have assigned a definite function to this organ. Because the maximum development of the tonsils coincides with the period in childhood of greatest increase in height and weight, there has been assigned to these lymphoid masses the rôle of a defensive mechanism. Against the statements of the pathologists on the defensive rôle of the tonsils is the evidence presented by clinicians, based on controlled studies, which does not always support the theory of defensive mechanism.

Clinicians, however, cannot be unmindful of the fact that the tonsils play some part in the defensive mechanism of the individual and especially in the young child. Just how this defense mechanism works and when it fails to work is not clearly understood. When once the factors involved in the defense mechanism are thoroughly known our present attitude to the tonsils may be altered. Of the various functions ascribed to the tonsils the cytogenetic function and the one that suggests that healthy tonsils help to confer immunity by protection against bacterial invasion seems most plausible. In defense of the cytogenetic function theory it has been demonstrated that white corpuscles can migrate from the lymphoid tissue of the tonsils into the crypts between the stratified epithelial cells — thence into the

pharynx, where they destroy micro-organisms. However, when one considers the studies of Pilot and Davis who were able to demonstrate that the incidence of hemolytic streptococcus was reduced about 50% following tonsillectomy it seems highly improbable that the theory of cytogenetic function is justified. There is some evidence to support the theory that healthy tonsils help to confer immunity by protection against bacterial invasion. It has been shown that the lymphocytes in the tonsils are continually migrating through the epithelium phagocyting the bacteria into the crypts and returning into the lymph-nodules where the bacteria are destroyed and some measure of immunity attained. This protection as a first line of defense is a function of healthy tonsils only and during the earlier years of life. It is suggested that their associated cervical glands act as the second line of defense. Clinical experience seems to support this contention that in a young child under six years of age healthy tonsils have a protective function. Our own statistical observation showed that the routine removal of tonsils, known to have produced no clinical symptoms prior to the operation, tended to make those children more susceptible to acute infections. Where the tonsils were not removed in children free from complaint there was less evidence that these children became more susceptible to infections over the same period of years.

Even if one accepts the theory that the lymphatic tissue in the nasopharynx contributes to what we term the development or maintenance of immunity, the mechanism involved is not understood. In spite of this vague and unconvincing evidence regarding the physiology of the tonsils there are very positive reasons for the surgical removal of tonsils. Clinical experience and controlled studies of children have shown that the tonsils causing mechanical obstruction and the tonsils presenting evidence of infection are an adequate indication for enucleation. Any interference with normal deglutition and breathing especially in the supine position handicaps the development of the child. As a rule, examination of the nasopharynx determines this impediment, but at times improper decisions are made following examination of the throat unless the complaints reported by the parents are given due consideration. Not all large or hypertrophied tonsils are causing mechanical obstruction. They may be causing a child not the slightest discomfort nor in any way retard the child's development. On the other hand, marked

hyperplasia of the tonsils need not be evident to account for an obstructive factor. Due evaluation must be given to the subjective complaints and that data added to the results of the physical examination forms a reasonable criteria for tonsillectomy or for the decision against such a procedure.

Few physicians in any field are in disagreement as to the advisability of removing infected tonsils but to obtain agreement as to when a tonsil is infected is a more difficult and uncertain matter. The pathologists have given us a fairly accurate description of a diseased tonsil but unfortunately to utilize their data one must see the tonsil in section under the microscope. According to the pathologists a diseased tonsil shows marked increase in the lymphoid tissue: lesions in the crypts, desquamation of epithelial lining, plugging of the orifices, dilatation of the lumen into cyst-like spaces, and occasionally the formation of minute abscesses. Pathologists are prone to consider the mere presence of purulent débris and bacteria in the crypts a normal condition of tonsils. The presence of epithelial abrasions, ulcerations, minute abscesses and the existence of polymorphonuclear leucocytes within the crypts constitute more definite signs of disease. Inasmuch as it is impossible to detect these epithelial changes except in the grossly infected tonsils it becomes necessary for the clinician to utilize other methods of determining when a tonsil is infected. The clinician may recognize these abnormal conditions in a tonsil which will aid in making the decision of a normal or a diseased tonsil:

(1) The escape of pus from the crypts when pressure is applied to the tonsil. (2) An enlarged tonsillar gland behind the angle of the jaw, varying in size and sometimes tender. (3) A purplish red tinge limited to the anterior faucial pillar. (4) Abnormal preponderance of leucocytes in material from the tonsillar crypts, frequently accompanied by general hyperemia of the pharyngeal and palatal mucosa. Cultural studies of the tonsils are helpful in making the diagnosis of diseased tonsils where specific infections are suspected. This is particularly true where the diphtheria bacillus, tubercle bacillus and certain fungi are isolated from the tonsils. The common bacterial invaders of the throat usually found on surface cultures or even from the crypts of the tonsil do not necessarily indicate a clinical infection from such an organism. A statistical study of cultures from normal and from diseased throats show that 50% of apparently

healthy tonsillar crypts harbor the streptococcus hemolyticus. It appears that this organism is more often present in the tonsils of children under twelve years of age than among adults. Likewise, the bacterial flora of the tonsillar surface and crypts varies with the geographical location of the individual and with the prevalence of infection at various seasons in the community.

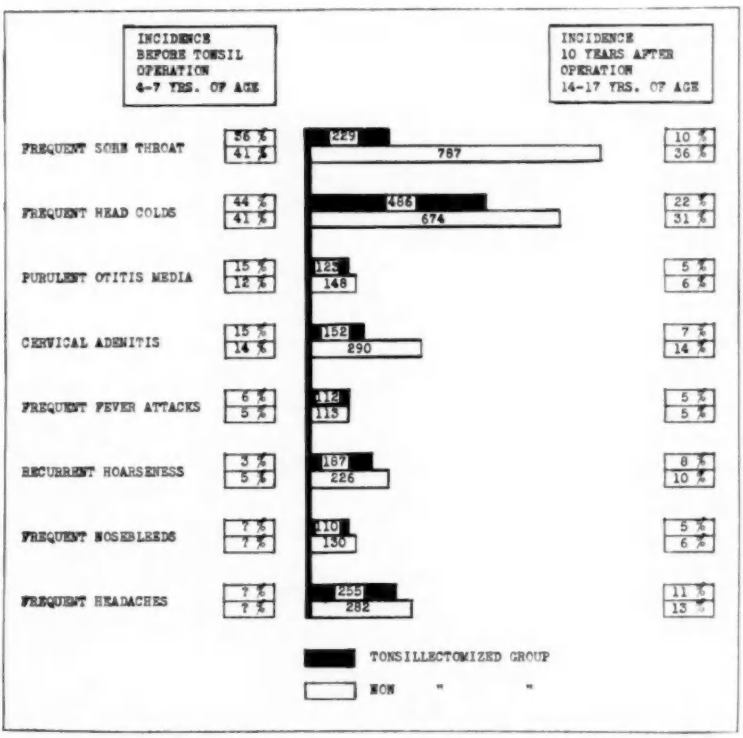
Utilizing whatever help may be gained from cultural studies of suspected tonsils and interpreting the pathological changes one can recognize by careful examination of the tonsils, the decision as to whether tonsils are diseased must be influenced by the symptomatology presented in each case under consideration. As with the diagnosis of appendicitis and gall bladder disease where the decision of operation usually rests with the clinical judgment based on the symptomatology and physical examination, so the case of the tonsils must be considered. It is here where honest difference of opinion is noted, for the sequence of events in the course of numerous infections is not thoroughly understood, so naturally the part played by the tonsils in these infections is undetermined. Basically it can be assumed that the tonsils may cause repeated infection in the throat and in the lymphatic drainage system. There can be little doubt about the fact that tonsillitis, peritonsillar abscess and cervical adenitis are usually due to an infection in the tonsils. It can, likewise, be assumed that certain systemic diseases have their origin in the tonsils. There is also very definite reason to believe that the tonsils may harbor an infection of a type that neither produces the well-known local clinical manifestations nor the classified systemic infections. Sufficient toxemia may be present in such children to impede the normal growth processes or to influence unfavorably certain physiological processes such as appetite, sleep and digestion. It is true that the relation of infection in the tonsils to any of these pathological states varies in different climatic areas and in different races. In tropical and subtropical countries the tonsils seldom become infected in children and consequently the tonsils are rarely incriminated where certain disease states are noted, but in the temperate regions where the tonsils along with the mucous membrane of the nasopharynx are constantly exposed to infections the tonsils are more likely to be involved in the disease cycle.

Granted that careful consideration is given to the symptomatology in every case, the diagnosis of infected or normal tonsils

will not always be certain. The same may be said in the final verdict when appendix disease and gall bladder disease are being considered. Where reasonable suspicion exists the decision to operate is usually made and the same should be done in the case of diseased tonsils unless the reasons for deciding against the operation outweigh the benefits to be derived from tonsillectomy.

Various attempts have been made to define definite indications for tonsillectomy. The suitable age period has been mentioned, even the season of the year best suited for the operation has been suggested. Tonsils have been grouped according to size and appearance. It became the practice in school and clinic examinations to recommend tonsillectomy largely on the appearance of the tonsils and little or no consideration was given to

CHART I



Incidence of complaints in 2,200 children before tonsillectomy and ten years after operation as compared with an equal number of controls.

the clinical history of the patient. In more recent years less importance was attached to the appearance of the tonsils than to the history of infections. Common sense is gradually supplanting arbitrary rules in deciding when the tonsils should come out and clinical judgment in the future will be influenced by the careful studies and end results noted in tonsillectomized children where liberal indications were accepted.

Already a number of indications that were accepted ten years ago as quite certain are today considered questionable. For example, it was taught that the removal of the tonsils after an attack of rheumatic fever would be quite likely to prevent a recurrence of such an infection. Today adequate evidence is at hand to show that such is not the case. With due consideration to recent reports on the immediate and late effects of tonsillectomy one can construct certain definite indications for tonsillectomy in children which should aid the clinician for the present.

In addition to the mechanical factors which serve as a definite indication for tonsillectomy there are certain infections and complaints that exist at present or are known to have existed in the child. Repeated attacks of tonsillitis even in children as young as two years of age are an indication for tonsillectomy. Certainly one or two attacks of this disease when a good recovery is made is not sufficient reason for removing the tonsils. Closely associated with tonsillitis are attacks of cervical adenitis. Where it can be shown that the repeated attacks, or a chronic attack, of cervical adenitis are not secondary to infected teeth it is desirable to remove the tonsils. Not only are about one-half of the tonsillectomized children with this indication relieved of a recurrence of a similar complaint, but undoubtedly a certain small percentage are spared a systemic infection such as rheumatism and nephritis that eventually may be a sequela to repeated throat infections. The presence or history of otitis media serves as a doubtful indication for tonsillectomy. It was found that primary attacks of purulent otitis media occurred only slightly less often in non-tonsillectomized children than in those whose tonsils had been removed. Where the tonsils and adenoids were removed to prevent recurrent attacks of otitis media it was shown that no appreciable difference existed in the operated children from the control group whose tonsils and adenoids were not removed. Children between the ages of one and three were the exception for they are definitely benefited after the

operation. Undoubtedly obstructive or infected adenoids are chiefly responsible for the tendency to ear infection in young children and the tonsils have less to do with ear infections.

The complaint of frequent head colds, common to many children, has often served as an indication for tonsillectomy. Certain children are benefited as far as this infection is concerned following tonsillectomy but statistically it has been shown that relatively few children are helped in escaping this type of infection. A history of frequent head colds must be considered a doubtful indication for tonsillectomy. Adenoidectomy alone will usually give greater relief than removal of the tonsils.

Other complaints such as unexplained attacks of fever, recurrent attacks of hoarseness, nosebleeds and headaches have been attributed to diseased tonsils. Tonsillectomy has been performed in many instances to relieve these complaints and prevent their recurrences. A critical study of the end result of tonsillectomy on these complaints showed that the tonsils are probably not responsible for these clinical complaints in many children. Children subject to recurrent attacks of hoarseness whose tonsils were removed showed a lesser incidence of this complaint than a similar group whose tonsils were not removed for this complaint. The same result was noted in children where headache was a complaint prior to tonsillectomy. Where unexplained fever attacks and frequent nosebleeds existed, the tonsillectomized children showed no advantage over the unoperated children with similar complaints.

Systemic infections in many instances may have their inception in the tonsils and may demand the removal of the tonsils at a proper time. On the other hand today it is generally accepted that rheumatic heart disease and nephritis may have no relation to a tonsillar infection. In fact such infections may develop years after a child has had a thorough tonsil enucleation. However, unless one can positively demonstrate that the teeth, the sinuses or some other organ serves as the focus of a systemic infection, it is wise to suspect the tonsils. When one surveys the value of tonsillectomy in the treatment of systemic infections judged from statistical studies the results are discouraging. There are, however, many excellent clinical results which justify this procedure even if uniformly the results are not good.

Several careful studies have been made on the relationship of the tonsils to the rheumatic syndrome. In general, there is agreement in the results obtained. Primary attacks of rheumatic disease, notably rheumatic fever, occur somewhat less frequently in children whose tonsils have been removed. Chorea seems to be less influenced by the presence or the absence of tonsils. With less rheumatic fever in operated children, rheumatic carditis is less common. A recent critical survey of 1,200 rheumatic children showed that the mortality was twice as great in rheumatic children whose tonsils were still in compared with those whose tonsils had been removed prior to the rheumatic infection. The removal of the tonsils after rheumatic disease had manifested itself did not prevent recurrence, for the unoperated children fared as well as the tonsillectomized children. At present it seems desirable to give the child the slight benefit that tonsillectomy offers the rheumatic child. If some of the more serious manifestations of rheumatic disease can be controlled even though recurrences of a milder nature occur the operation is justifiable and desirable.

Further observations on the effect of tonsillectomy on other infections showed a definite decrease in the incidence of diphtheria among the tonsillectomized children. Scarlet fever to a lesser degree was controlled by removal of the tonsils. The complications of scarlet fever were less severe in the tonsillectomized children. Measles existed with the same frequency and severity whether the tonsils were present or absent.

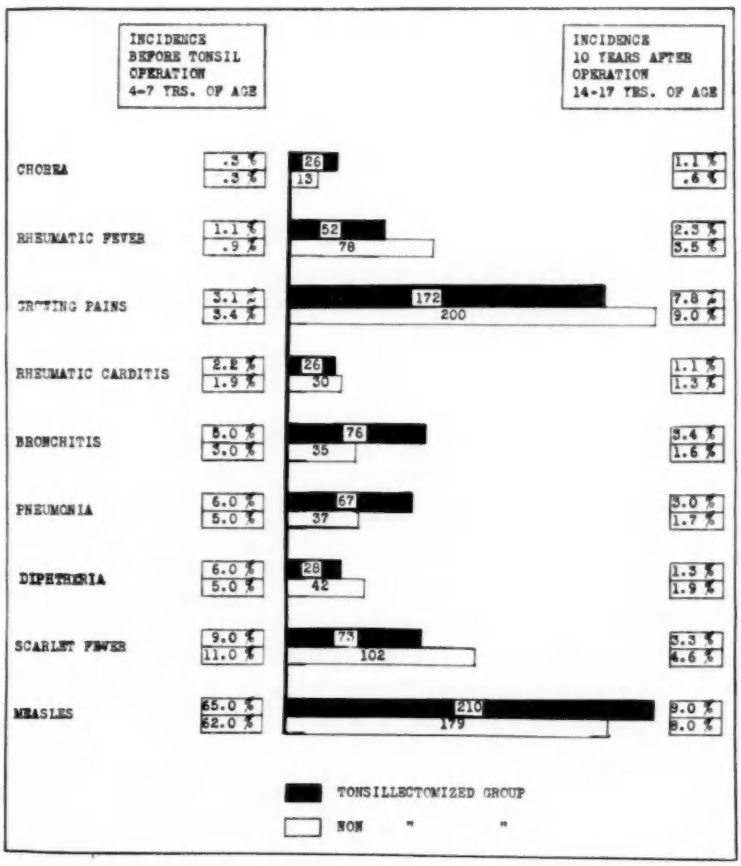
The benefits noted in diminishing the incidence of upper respiratory infections after the removal of the tonsils were not evident in surveying the occurrence of lower respiratory infections. In fact, bronchitis and pneumonia occurred with greater frequency in tonsillectomized children than in those whose tonsils were not removed.

Long standing anorexia, malnutrition, cyclic vomiting and chronic fatigue may be evidences of a low grade chronic infection originating in the tonsils. These complaints, however, may be due to other causes for these same symptoms are found in children whose tonsils have been thoroughly enucleated. Before accepting these complaints as indications for tonsillectomy all other possible causes should be considered. If none can be found it is fair to assume that an infection in the tonsils may be responsible and an operation is not only justifiable but desirable.

It is quite evident that a positive diagnosis of infected tonsils cannot always be easily made. Too little effort has been made to reach a fair decision as to the part that tonsils play in local and in systemic infections. Consequently, the tonsils have been frequently removed without a real indication. Naturally, no real benefit to the child can accrue in such instances.

There are definite contraindications for tonsillectomy which should be heeded even when the indications appear positive. Where hemophilia, leukemia, severe anemia or Hodgkin's dis-

CHART II



Incidence of infections in 2,200 children before tonsillectomy and ten years after operation as compared with an equal number of controls.

ease is known to exist no tonsil operation should be undertaken. If thymic disease is suspected no operation should be performed without an x-ray of the chest. In the presence of or immediately after the acute infections such as acute head colds, acute tonsillitis, acute otitis media, acute bronchitis and acute nephritis removal of the tonsils is not advisable. An extension of the infection or a septicemia may complicate the operation in such cases. In acute rheumatic heart disease no good can be accomplished by tonsillectomy and further embarrassment of the heart may result. Experience has shown that in acute chorea an operation on the tonsils is likely to intensify the symptoms, so it is desirable to defer the operation until the acute symptoms of chorea have subsided.

Though the hazard of removing the tonsils in children with some of these contraindications is small, it exists more certainly than in children without them. Even under the most favorable conditions unexpected complications occur so that at no time unnecessary risks should be taken.

The relation of the tonsils to the health and growth of a child has by no means been settled. A decade or two ago little opposition was found to the opinion that any child is better off without tonsils. Tonsils were considered a breeding place for infection and if not already in that state would be so in a few years. It seemed as though there was no sound argument against universal or prophylactic tonsillectomy. It has developed, however, in the last years, that tonsillectomized children did not escape in many instances the infections which were prevalent in a community. Some children who had little or no disease were actually having more trouble than before the tonsil operation. It was noted that deep respiratory infections such as laryngitis, bronchitis and pneumonia actually occurred more frequently in tonsillectomized children than in control children. Account also had to be taken of the rare operative death due to hemorrhage or to the anesthesia used and to unwished for lung abscesses following tonsillectomy. The immediate benefits enthusiastically reported by parents were not lasting and when unoperated children were observed over a period of years with the operated group it was found that some of the benefits credited to the operations were common to both groups. These clinical and statistical findings have brought about a changed attitude to the tonsils and their treatment. The surgical treatment of the ton-

sils has developed to a high degree. The best technique assures a complete enucleation with a minimum of shock and hemorrhage to the child. Recent statistical studies on the end results of tonsillectomy have influenced some physicians to underestimate the importance of the tonsils as a factor in disease. The over-enthusiasm of a few years ago with its consequent disappointments has been somewhat responsible for this state. The outstanding problem confronting the physician is to discover a more accurate way of diagnosing a diseased tonsil. There is no known therapeutic procedure that is of greater value to a child than the removal of diseased tonsils. The failures are largely due to a wrong diagnosis. When once a better selection of candidates for this operation is made the effectiveness of the operation will be accepted.

More information on the factors that are involved in local tissue immunity is needed. It might be mentioned that experiments with products of hemolytic streptococci for skin testing are being made to determine if children with infected tonsils respond differently from those with tonsils presumably not infected. Likewise, studies on the vitamin content of tonsils are under way which may have some significance. Until the investigator comes to the assistance of the clinician the old method of selecting the candidates for the removal of tonsils must continue.

SYMPOSIUM ON TONSILS

TONSILS AS FOCI OF SYSTEMIC INFECTION

H. Archibald Nissen, M.D.
(By Invitation)

BOSTON

Recognition of a problem is the first step toward its solution. The problem to be discussed is: *Tonsils as Foci of Systemic Infection.* In order that the words "tonsils," "foci" and "systemic infection" may convey the same meaning to each of us, they must be defined.

1. *Tonsils*, the pharyngeal and the lingual. These represent specialized lymphoid tissue. However, any tissue of the same composition, even if not unified structurally with separate vascular-nerve supply, must be considered as important in its relationship to direct infection or allergic reaction, and the transmission of infection probably through lymphatic channels. In other words, the nasopharynx contains a great deal of tissue which is very similar in composition. Some is called lymphoid tissue, and the more specialized tissue, tonsil. Therefore, the word "tonsils" means *lymphoid tissue*, other than nodes, above the seventh cervical vertebra.

2. *Foci.* A focus of infection is understood as a locus containing bacteria or inflammatory tissue, with or without the constant presence of bacteria ascertainable.

3. *Systemic Infection.* First, the entire body may be involved. Second, various systems of the body, such as the cardiocirculatory, genito-urinary, bones or joints, etc., may be specifically or singly involved.

4. The problem restated then is: Can the lymphoid tissue located above the seventh cervical vertebra be a focus, or trans-

mitting agent, for the production of infection or disease in one or more systems composing the body structure? Is there immediate, delayed, or constant response to acute, subacute or chronic inflammatory change in the lymphoid tissue?

It is difficult to prove that such infection is the activator or producer of the systemic change in the body, but, if proved, extirpation of this source of infection logically follows and with extirpation halting the progress or advance of any systemic disorder which has depended for existence on the intermittent or continuous manufacture of toxic irritating factors, either by direct transmission or by the production of an allergic reaction in the other tissues of the body.

Obviously, we would not be here today discussing these problems had Science, which means measurement and fact, found the answers. Training, practical experience, long time observation and knowledge of the patient are all needed; and the *same* group of patients must be followed and observed for several years to prove clinical association between nasopharyngeal infection and systemic reaction.

In my experience there are four types of tonsillar and nasopharyngeal reactions indicating infection which may affect systemic disorders.

(1) There is the acutely infected tonsil, with and without abscess formation. This is easily recognized.

(2) There is the red, hypertrophied chronic tonsil with and without symptoms. This also is easily recognized and is obviously infected. However, the individual possessing such tonsils is often completely free from any subjective symptoms and is unaware of any relationship, subjective or objective, between his throat and systemic disease when present. There is constant absorption from such infected foci, though the toxic or bacterial activity may be of a mild low-grade type.

(3) The third reaction is not easily recognized, and plays a most important part in connection with systemic response to its toxic irritants. In this reaction, the anterior pillar, particularly the portion overlying the upper half, is a purplish red color, as

are the edges of the soft palate, and at times the uvula. If this throat is observed closely from time to time, the appearance will be seen to change from the dull, purplish red to a much more brilliant inflammatory shade, but with the underlying purplish tinge remaining, and becoming more pronounced. This is the type of infection in which a sore throat is rarely mentioned, unless to it is added exogenous infection. Exogenous infection means an infection entering the body from without; endogenous infection, one which is carried in the body. The difference between exogenous and endogenous infection is in the *duration* of the changed appearance of the throat. In endogenous reaction, with or without systemic response, the tonsillar picture subsides quickly and returns to its former chronic appearance in a day or so; the systemic reaction, if recognized, is also of short duration. Exogenous reaction is actually a subacute tonsillitis, or an acute exogenous infection superimposed upon a previously chronically infected, or functionally destroyed, tonsil.

This type of tonsil may have retrotonsillar adhesions, abscesses, or, what is more likely, under the congested discolored anterior pillar and soft palate, a single large abscessed region, or numerous small pockets, which may or may not open to the surface. This is concealed in a cursory view of the throat, but if the anterior pillar and soft palate are separated, it will be seen. This type of tonsil is of particular importance because it may be a transmitter of direct infection or may produce allergic reaction on the part of the body without subjective sore throat. (This throat differs from the well-known "smoker's throat," which shows obvious pharyngeal, rather than tonsillar, irritation).

My attention was first directed to this type of nasopharyngeal infection in 1918 when I was called to see a woman of sixty, complaining of brilliant red, swollen, painful distal phalangeal joints; with moderate fever, but no sore throat. These joints had heretofore showed degenerative changes but without symptoms. Obviously some additional factor had produced this change. Though subjective throat symptoms were denied, local examination showed two small, brilliant red spots in the tonsillar fossæ (the tonsils had been removed years previously). Culture from these spots showed a growth of streptococcus only. Within ten days the throat had resumed a perfectly normal appearance to my then-inexperienced eyes. The small red areas were no longer visible. Coincidentally the joints had also subsided. Later

the same sequence of events was noted. Consultation with various otolaryngologists followed. Each reported the throat as negative for infection, or as a source of whatever had activated the degenerative joint changes to acute reaction. Eventually, a consultant saw the patient when the throat was again at the height of a flareup, again without local symptoms, but with the same joint response. The same red tabs were observed on top of a dusky purplish area. During the next period of quiescence these tonsillar remnants were removed, and for the remaining ten years of her life the patient experienced no further joint flareup.

Similar clinical observations were made in the ensuing years. In 1919, a series of patients with and without tonsils suffering from various chronic diseases was studied. The group showed from time to time a flareup of systemic disorder, with moderate fever of a few days' duration, and, if arthritics, definite joint reaction; but *no* subjective throat symptoms. In each instance, examination of the throat showed this same marked discoloration of the membrane above the tonsil, that is, the anterior pillar, soft palate and uvula. According to the short or prolonged duration of the flareup the reactions were considered as of endogenous or exogenous origin. Fatigue, loss of sleep, emotional upset, etc., were some of the factors which produced systemic body reaction to this latent tonsillar infection.

During the period of quiescence these throats had the appearance of what I term chronic tonsils, but which were not sufficiently obvious to interest the average otolaryngologist until he happened to see the same throat during its active stage. In this group of patients the tonsils had been considered, and dismissed, as playing a part in the production of recurrent joint flareups; but after repeated observation, and the gradual recognition of this particular nasopharyngeal reaction, it was agreed that the tonsils did play a part in the systemic disorder. They were removed during periods of joint and throat inactivity with gratifying results in a certain proportion of the patients. This third type of tonsillar reaction will be recognized only if the patient is watched day by day for some weeks, and sometimes for months. It will then become evident that this discoloration in the tissue subsides, disappears, and reappears; but at all times there remains the underlying picture of chronic infection. Without repeated examination it often will be overlooked, ignored or misinterpreted, though it is an actual factor in the production or maintenance of chronic systemic disease.

(4) The fourth type of infectious lymphoid tissue in the throat is found in the nasopharyngeal cavity itself, which is very often a greater source of infection than is realized. If it is not examined repeatedly, as in the third type of throat, it may be considered as normal or of no importance. It is due to this type of tissue that certain tonsillectomized patients continue to have their former complaints of sore throat, colds, etc., with the same systemic reaction. Many of these individuals come for treatment of a severe head or chest cold, denying any sore throat in connection with the infection. More careful questioning may reveal a mild scrapy throat of such short duration, and so immediately followed by acute involvement above or below that it had been forgotten. The nasopharynx, however, had certainly been the original host to the invading bacteria. This lymphoid tissue may well be considered as small atypical tonsils, subject to exactly the same infectious changes as the tonsils, and probably responsible for much the same systemic reaction. Tonsils when infected may be compared to lobar pneumonia; nasopharyngeal lymphoid tissue, other than the tonsils, when infected, to bronchopneumonia, or many little pneumonias.

Cultures from the nasopharynx during disease of infection show staphylococci more frequently than they do any other type of bacteria, so that it has become almost axiomatic that nasopharyngitis in chronic disease is treated as a definite staphylococcal infection. On the other hand, true tonsillar infection cultures show mixed bacteria, or more frequently streptococci. Cultures taken at different stages of an acute tonsillitis may show a complete cycle of the streptococcal bacteria beginning with a non-hemolytic streptococcus, and winding up again with the non-hemolytic. Whether the staphylococcus is a pleomorphological bacterium has not been proved satisfactorily to date. The direct effect of the nasopharyngeal infection, most frequently staphylococcic, in chronic disease has not been given sufficient consideration.

Shortly before leaving Boston I was very much pleased to find that twenty or thirty years ago Dr. Mosher had, at that time, from clinical observation and some culturing, reached or suggested the same point of view that I am presenting here regarding the potential or probable staphylococcic pleomorphology.

SYSTEMIC INFECTION FOLLOWING NASOPHARYNGEAL TISSUE INFECTION

Recognition of infected nasopharyngeal lymphoid tissue must be followed by recognition of its influence upon the rest of the body. Clinical observation and years of practical experience have proved to the satisfaction of most clinicians what science has failed to prove as yet, i.e., that infection in the nasopharyngeal structures has direct bearing on changes in the respiratory tract, the gastro-intestinal tract, the genito-urinary tract, the skeletal structure, and the homeostasis of the body. Whether the first attack of tonsillitis ruins the functional activity of that tonsil, with or without retention of infection, is in a way beside the point, because other lymphoid tissue in the throat may be the transmitter of direct infection or the sensitizing agent for allergic reaction in other body systems.

Infection directly transmitted follows the lymphatic course forming depots in the chest with hilus gland involvement, which, in turn, if inadequate in their function, produce local or diffuse distribution of infectious material. At any stage a few, or many toxic elements, or bacteria, may enter the bloodstream producing a definite bacteremia of minor or major degree. Also at times a localized abscess may be formed which without proof of bacteremia may serve as a source of allergic response on the part of the body. Such response may be in the form of well recognized skin reaction, — urticaria or hives, — angioneurotic edema, "hay fever" (vasomotor rhinitis), or possibly joint reaction of a type which may be termed hay fever in a joint. Allergic symptoms manifest themselves in two ways: first, spasm of smooth or involuntary muscles; second, vascular disturbance, probably increased capillary permeability.[1]

It is imperative, therefore, that intensive individual study of each patient be made; that the otolaryngologist have at his command the assistance of the laboratory, roentgenologist and clinician in order that the optimal time for operative interference be chosen. Unsatisfactory results of operation, though they may be in the minority, have lessened, unfortunately, general interest in infection above the seventh cervical vertebra as a causative agent, or transmitter of infection, affecting the rest of the body. My own experience has proved to me that infection in the nasopharyngeal tissues has a definite effect on systemic disease; that in some instances it should be removed; in others it should not;

that there is a time in each patient's course when operation will prove of definite benefit; that there is also a time when it will prove disastrous. The mere recognition of nasopharyngeal infection, and the fact that it is definitely an extra load which has precipitated its host into some other systemic infection, caused a recurrence of a previously quiescent disorder, or pushed the patient over the borderline into a downhill course, is not of *itself* sufficient to warrant operative interference. Intelligent and successful choice of the time for operation depends upon study of each individual patient, and should include:

1. A history that is complete in every possible detail.
2. A complete physical examination as well as local examination.
3. Examination of the blood, — the red cells, white cells, hemoglobin, smear, sedimentation index. This will rule out any actual disease. Removal of infected areas during the height of activity of any systemic disease, especially during the acute inflammatory condition of joints in arthritis, is more likely to be followed by disaster than triumph. A possible exception to this is in the rheumatic fever syndrome. Here even during the beginning or height of the acute condition tonsils are often better out than in. In time this may be proved true also for scarlet fever.
4. A seven foot plate of the chest to recognize arteriosclerotic or hilus changes, unrecognized rheumatic heart, tuberculosis, bronchiectasis, so-called chronic bronchitis, or emphysema. Any of these findings indicate another reason for treating tonsillectomy as a major operation, "not to be entered into lightly or ill-advisedly."
5. Examination of urine is indicated obviously for the presence of nephritis, pyelitis, hematuria, etc. However, it is so well recognized a fact that it is frequently forgotten, that a kidney may be much damaged and still produce apparently normal *single* specimens of urine. Therefore, dilution and concentration tests, phthalein output, etc., are as important pre- and post-operatively in tonsillectomy as in any other major operation. These tests indicate the acual physiological function of the kidney. Excellent detailed descriptions are given in Fishberg's recent book on hypertension.[2]
6. A clinical observation which should be given serious consideration is marked tissue change in a patient. Tests of all

kinds may be within normal limits, and the only outstanding index of physiological dysfunction may be marked loss of weight, — thirty, forty or fifty pounds in one year, — without recognized systemic disease. If tremendous loss of weight is combined with other systemic disorders, it must be considered as a definitely poor prognostic sign, especially in an individual in whom all forms of nutritional upbuilding have failed.

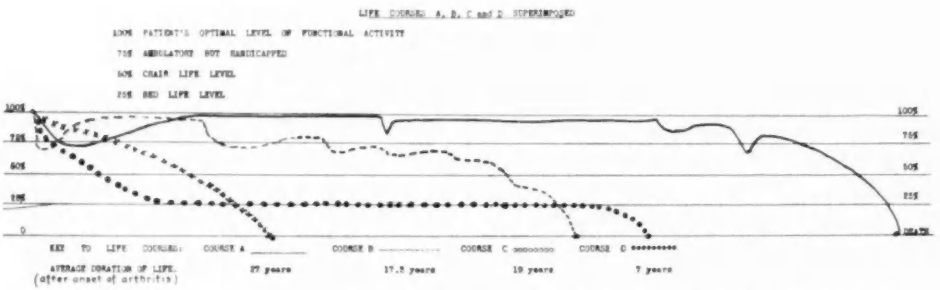
7. If this complete checkup of each patient is more than the otolaryngologist has time to carry out, then as the laboratory is used for its help, so the internist should be used; and the combined knowledge of both be brought to bear upon the problem. Often the one cannot work independently of the other.

8. Lastly, if, and when, tonsillectomy has been performed, each patient should be followed and watched just as carefully *after*, as he should be before, operation. Only in this way will it be learned whether the removal of the source of infection was of demonstrable value to that patient, whether it halted the progress of his systemic disease, or whether it added an additional load which he was unable to carry and so handicapped him further. Careful study of the individual, both before and after operation, is necessary if definite relationship between tonsils and systemic infection is to be *proved;* and the value of surgery versus conservative treatment is to be intelligently demonstrated.

My former ideas on this subject have been confirmed in the past few years by an intensive study of a group of about five hundred arthritic patients.* Arthritis is a disease, (and in my opinion, *one* disease, though years have been devoted to fruitless discussion of *types* of arthritis) which is the epitome of all chronic disease. A group of arthritics represents the best possible single cross-section of general systemic dysfunction. This study covered, first, a group of ninety-five who with death

* The majority of these patients were followed at the Robert B. Brigham Hospital in Boston from 1914 to January, 1934. The coöperation of the staff of the hospital enabled me to study the records and check on the patients. The assistance of Dr. Charles T. Porter, Chief of the Otolaryngological Service, was of particular value. He gave generously of his time and knowledge, so helping to complete the picture of the individual patient.

had completed a cycle of chronic disease.[3] Later a group of four hundred living were similarly analyzed. These people, regardless of age, duration of arthritis, or other systemic complication, followed four distinct courses from the cradle to the grave; and I believe all chronic patients would show similar life courses were they charted in like manner.



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FOUR LIFE COURSES SUPERIMPOSED ON ONE ANOTHER

Course A represented a patient who with the onset of arthritis dropped to a lower level of activity, remained there for a time, then climbed back toward his former level and stayed at this level through the greater part of his remaining life. Course B showed a life course made up of definite periods of remission and relapse, and as the years passed, the trend gradually downwards, with lengthening and more frequent periods of relapse and shorter and fewer periods of remission. Course C showed a tremendous drop in the life curve, over a variable period of time, from the expected 100% of activity to a 50% or 25% level, because of extensive joint destruction and crippling; and the remaining life spent at this low level, often for years. Course D showed a course which progressed steadily downward to death, without appreciable remission, regardless of treatment or any one particular factor.[4]

A detailed study of this group of arthritics from the point of view of the nasopharynx as a source of infection showed certain outstanding facts.

At the Onset of Arthritic Symptoms
10% had had tonsillectomy performed
90% *had retained their tonsils intact*

Is this ratio of tonsillectomized patients to those with retained tonsils merely a normal incidence?

Does it indicate on the part of the average physician lack of recognition of chronic nasopharyngeal infection, particularly of the third and fourth types described?

Or does it indicate that a physician was given no opportunity to decide whether the nasopharynx was or was not a source of infection? The average patient in a public hospital ward is usually one without means, often with no great intelligence, and from a social stratum where such relatively minor body ills as repeated colds, repeated attacks of sore throat or tonsillitis occurring yearly or even more frequently are taken as part of the "day's work," and are allowed to continue without medical care. It is only when something new and incomprehensible is added to this accepted condition that medical aid is sought. By that time the spread of nasopharyngeal infection through the system has been accomplished, and in all probability it is too late to gain a great deal by removal of tonsils though they may have been a *source* of systemic infection years before.

In any case the mere fact that in a group of nearly five hundred arthritic patients 90% *had* retained their tonsils intact up to the time of the arthritic onset (and this group included patients fairly equally distributed in each decade of life) arouses one's interest in the speculation as to whether the arthritic picture would have been the same had the tonsils been removed in early childhood before the first attack of acute infectious tonsillitis occurred. Might such procedure have avoided future tonsillitis so lessening an extra load on other lymphoid tissue in the nasopharynx? Might it later in life have eliminated the possibility of tonsils being examined and considered as innocuous though in reality they were a definite source of infection missed by the examining physician because of lack of symptoms or complaints on the patient's part and lack of repeated observation of the nasopharynx on his own part?

TABLE I — *Entire Group of Arthritics*

Life Courses	T. & A. Before Arthritic Onset	Tonsils Intact at Arthritic Onset
Course A	17%	83%
Course B	7%	93%
Course C	11%	89%
Course D	18%	82%
Group followed 5 years or less	17%	83%
TOTAL GROUP	10%	90%

TABLE II — *Tonsils Out Before Arthritic Onset*

Percentage Showing Recognized Joint Reaction to Nasopharyngeal Infection

Life Course A	20%
Life Course B	25%
Life Course C	66%
Life Course D	100%
Group followed 5 years or less	50%

The actual numbers in the above group were small (5, 14, 6, 2 and 10), but the *disproportion of the results* made the observations worth noting.

TABLE III — *Tonsils Intact at Onset of Arthritis*

50% had had recognized nasopharyngeal infections before and during their arthritis.

45% had TONSILLECTOMY AFTER the onset of arthritis.

55% have retained TONSILS INTACT up to the present.

TABLE IV — *Tonsils Intact at Arthritic Onset*

Life Courses	Tonsils Retained to Date	T. & After Arthritic Onset
Course A	50%	50%
Course B	53%	47%
Course C	59%	41%
Course D	61%	39%
Group followed 5 years or less	59%	41%
TOTAL GROUP	55%	45%

TABLE V — *Tonsils Intact at Onset of Arthritis*

In COURSE C ONLY was there MARKED RECOGNIZED ASSOCIATION between joint reaction and nasopharyngeal infection.

Of the 29 patients in this COURSE C, or the 59% of that group who have retained their TONSILS INTACT to the present time, 41% showed this *definite reaction* of the joints to nasopharyngeal infection; and in half the patients the reaction was *tremendous* and repeated.

CONCLUSION

I am convinced that even without *recognized* association between upper respiratory tract infection and systemic (or more particularly joint) infection such association may be present. Proof of this depends on intensive study of the individual to determine:

1. Recognition of obvious and concealed chronic *infection in nasopharyngeal tissue.* The two most common types of infection are unmistakable, — the acutely infected tonsil and the red hypertrophied chronic tonsil. A third is possibly of greater importance, and is often missed unless the patient is seen frequently, and the appearance, subsidence and disappearance of the signs of infected lymphoid tissue are noted. On examination of this type of throat a purplish red discolor-

ation is seen on the anterior tonsillar pillar, along the edges of the soft palate and at times in the uvula. These local changes are constant to a certain degree (the dusky discoloration being present at all times), but show varying reactions to exogenous and endogenous infection. During such reactions the tonsillar poles become prominent and more inflamed, but this is noted only if the upper part of the tonsil is exposed. The duration of the reaction is short if the excitor is endogenous; long if of exogenous origin.

2. Recognition of single or multiple *systemic infection.* This depends on thorough physical, laboratory and x-ray examinations.

3. Determination of *association between local and systemic infection* by extensive and prolonged study of each individual patient.

Long observation indicates that Man follows, roughly, four natural life courses from birth to death. Study of a large group of patients with various chronic diseases has shown them to follow four life courses similar to the natural course, but more exaggerated in their variations. These life courses have been termed Courses A, B, C, and D. An individual following Course A, after a period of lowered functional activity, returns to nearly his former level, and remains there practically until death. One following Course B, from the onset of his disease until death, has periods of remission and relapse, with the life curve gradually sloping downward. A patient in the Course C group, with the onset of his disease, drops in a variable period of time to a definitely lower level of activity, and remains at this low level until death. In Course D, the individual's life course is comparatively short and the line shows a steady downward slant.

The ability to estimate the future life course of one's patient will prove of great value in determining the best procedure to adopt when nasopharyngeal infection is present. Generally speaking, if a patient presents himself with combined nasopharyngeal and systemic infection, having had no operation performed on the tonsils, I believe the lesser of two evils, — i.e., removal of tonsils as a possible focus of infection with the risk of disaster, or leaving them intact as a constant source of infection via drainage with equal possibility of disaster, — is REMOVAL. Suc-

cess or failure following such procedure depends on the TIME chosen for operation. If good chance (or good judgment) makes the time tonsillectomy is performed opportune, the result will be beneficial to the patient. This is the point on which attention should be focused, and more *certainty* gained as to this crucial time. Greater effort should be made to determine the optimal time for removal.

In an arthritic it should be performed when both the nasopharynx and the joints are as quiescent as possible; when the patient's resistance and general health have been raised to the optimal level; and *only* if the patient shows periods of such quiescence and measurable improvement. In an arthritic who appears never to reach even a short period of quiescence of joint disease, or of general improvement, who is markedly undernourished and who cannot be made to gain weight, I believe tonsillectomy is *never* indicated, and that the only practical way of keeping nasopharyngeal infection in check is by whatever local treatment can be used. This "hands off" policy is also indicated in any individual, whatever his disease, who is believed to be following Life Course D. Any operative interference on one in this group appears to be disastrous.

Wise decision between operation and conservative treatment of nasopharyngeal infection depends *wholly* on the INDIVIDUAL patient in question. Without a thorough knowledge of him and of his physiology the outcome is pure chance, and Science remains without proof of the relationship between tonsillar and systemic infection.

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SYMPOSIUM ON TONSILS

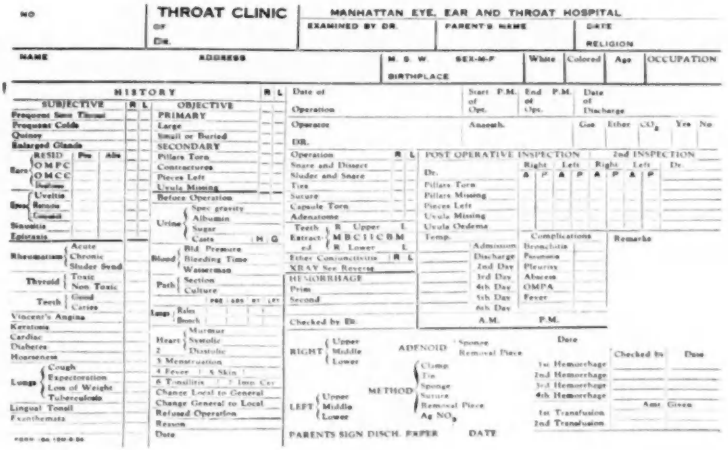
PREPARATION AND MANAGEMENT OF PATIENTS PREVIOUS TO AND DURING TONSIL OPERATION

DAVID H. JONES, M.D.
(By Invitation)

NEW YORK

THE PURPOSE of this paper is to tell what is being done in the Tonsil Clinic of the Manhattan Eye, Ear and Throat Hospital of New York City. The writer, from an extensive observation of work done in that institution and the benefits accruing to patients because of improvements in technique made in the last few years, believes it worth while, even though we all think we are competent to do that work, to let you know about the handling of patients both before and after the removal of tonsils and adenoids. Further, the author believes this operation should always be a hospital procedure.

May 1, 1929, the adenoid and tonsil work in this hospital was reorganized, being placed in charge of one surgeon. The first task was to secure a history card of a distinctive color enabling one to distinguish it readily from the nose and throat card. It was necessary that this card, with a minimum of clerical work, should give to the surgeon a picture of the patient both before and after operation. After two and one-half years' use the card, as originally designed, has proved satisfactory. The next problem was to select one surgeon from each of the four nose and throat clinics to carry on this work. After a trial of eighteen months it was found that the examinations and operations done by different men were not uniform, therefore, the adenoid and tonsil service, which is now functioning as a separate clinic was removed from the nose and throat clinics, and since its organization 58,750 operations have been performed to September 1, 1934.



NO

THROAT CLINIC

MANHATTAN EYE, EAR AND THROAT HOSPITAL

OF

DR.

EXAMINED BY DR. | PARENT'S NAME | DATE | RELIGION

NAME | ADDRESS | M. S. W. SEX-M-F | BIRTHPLACE | White | Colored | Age | OCCUPATION

HISTORY

SUBJECTIVE — R L

Frequent Sore Throat
Frequent Colds
Quinsy
Enlarged Glands
Ears { RESID Pus Abs; O M P C; O M C C; Deafness }
Eyes { Uveitis; Retina; Choroid }
Sinusitis
Epilepsia
Rheumatism { Acute; Chronic; Shoder Synd }
Thyroid { Toxic; Non Toxic }
Teeth { Good; Caries }
Vincent's Angina
Keratosis
Cardiac
Diabetes
Hoarseness
Lungs { Cough; Expectoration; Loss of Weight; Tuberculosis }
Lingual Tonsil
Exanthemata

OBJECTIVE — R L

PRIMARY
Large
Small or Buried
SECONDARY
Pillars Torn
Contractures
Pieces Left
Uvula Missing

Before Operation
Urine { Spec. gravity; Albumin; Sugar; Casts H G }
Blood { Bld. Pressure; Bleeding Time; Wasserman }
Path { Section; Culture }
Lungs { Rales; Breath { Murmur } }
Heart { Systolic; 2 Diastolic }
3 Menstruation
4 Fever 5 Skin
6 Tonsilitis 7 Intro. Cer.
Change Local to General
Change General to Local
Refused Operation
Reason
Date

Date of Operation | Start of Opr. P.M. | End of Opr. P.M. | Date of Discharge

Operator | Anaesth. | Gas | Ether | CO_2 | Yes | No

DR.

Operation — R L
Snare and Dissect
Sluder and Snare
Ties
Suture
Capsule Torn
Adenotome
Teeth { R Upper L; Extracted M B C I I C B M; R Lower L }
Ether Conjunctivitis — R L
XRAY See Reverse
HEMORRHAGE
Prim.
Second
Checked by Dr.

POST OPERATIVE INSPECTION | 2nd INSPECTION

Dr.
Pillars Torn
Pillars Missing
Pieces Left
Uvula Missing
Uvula Oedema
Temp.
Admission
Discharge
2nd Day
3rd Day
4th Day
5th Day
6th Day
A.M. P.M.

Right A	Right P	Left A	Left P	Right A	Right P	Left A	Left P	Dr.

Complications | Remarks
Bronchitis
Pneumonia
Pleurisy
Abscess
OMPA
Fever

ADENOID — Sponge, Removal Piece
RIGHT { Upper; Middle; Lower }
LEFT { Upper; Middle; Lower }
METHOD { Clamp; Tie; Sponge; Suture; Removal Piece; Ag NO_3 }

PARENTS SIGN DISCH. PAPER DATE

Date | Checked by | Date
1st Hemorrhage
2nd Hemorrhage
3rd Hemorrhage
4th Hemorrhage
Ans. Given
1st Transfusion
2nd Transfusion

(Front)

New York, 19

I, the undersigned, herewith give my consent to the Surgeons of the Manhattan Eye, Ear and Throat Hospital to perform such operations that in their judgment may be necessary, in the case of

Signed

Witness

RE EXAMINED

BY DR. DATE

1. DOES PATIENT HAVE LESS SORE THROAT?
2. IS PATIENT STILL A MOUTH BREATHER?
3. DOES PATIENT STILL HAVE FREQUENT COLDS?
4. HAS PATIENT GAINED WEIGHT?
5. PILLARS TORN?
6. PILLARS CONTRATURES
7. PIECES LEFT
8. UVULA MISSING
9. IMPROVEMENT

RHEUMATIC CONDITIONS

REMARKS

XRAY REPORT

(Back)

One surgeon is at the clinic daily from one to two o'clock examining all new patients. From two to three o'clock, if required, he operates on all patients under a general anesthetic, after which he returns to the clinic, where he finishes examining new or postoperative patients.

The first questions asked are: Why did you come? Have you been sick recently? If so, when and how long were you ill, and what was the trouble? Under subjective history it was found that 90% had suffered from frequent sore throat, viz., recurrent attacks of acute or chronic tonsillitis; 90% had had frequent colds, viz., an acute infection of the respiratory tract; 42% had enlarged cervical glands; 2½% quinsy; 8% deafness; 8% discharge from ears; 22% sinus involvement; 6% rheumatism; and 1½% cardiac disease.

All patients having recently recovered from any illness such as peritonsillar abscess, acute otitis media catarrhal, acute rheumatism, acute or chronic pulmonary complications, anæmia, hemophilia, diabetes, nephritis, and, in fact, any symptom or appearance which might complicate the operation, are referred to the medical clinic for a further and more complete physical examination and treatment. If examination shows no contraindications, patient returns to the tonsil clinic.

All adult patients are examined by indirect laryngoscopy. As it is impossible to do an indirect laryngoscopy on children, those with symptoms of hoarseness, as well as adults with laryngeal involvement, are referred to the bronchoscopic clinic for direct examination and consultation. Patients suffering from carious teeth are sent to the dental clinic.

Those with a history of syphilis have a Wassermann test, and if reaction is positive, operation is deferred. In a series of 110 colored patients (both sexes) it was found that 12 gave a strong positive reaction.

An xray of the chest, anteroposterior and lateral, is taken for enlarged thymus in children who show symptoms of any glandular involvement or dyspnœa. Our experience does not coincide with Dr. Mosher's* observations, namely, that 7½% of 2,344 children under ten years of age show enlargement of thymus.

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* Personal communication from Dr. Harris P. Mosher.

Urinalysis is required on all patients over three years of age, and a complete examination is done if albumin or sugar is found. Those with albumin are operated upon while those with sugar are referred to the medical clinic for further examination.

Under the heading "objective history" the surgeon states whether the tonsil is primary or secondary; under primary whether the tonsils are large, small or buried; under secondary whether the pillars are torn, missing, contracted by scars, pieces of tonsil which have been left, or uvula missing. The subjective and objective diagnoses are checked on the history card by a very efficient history clerk who also keeps a daily record of all conditions checked before and after operation. From these records monthly reports are filed in the record room.

NOTICE TO FEMALE PATIENTS

Do not accept a date for tonsil operation which falls within the five days previous to your monthly period or the five days during this period.

All adult female patients are given a printed slip stating: "Do not accept a date for tonsil operation within the five days previous to your monthly period or the five days during this period." It has been stated that the coagulability of the blood is lessened during the menstrual period, and our observations confirm this statement.

After a careful history has been taken and thorough examinations made by the various clinics to which the patients may have been referred, they are then sent to the office, where a date for the operation is given. Here the patient is advised to have his breakfast on the day of the operation and to return to the hospital at eight o'clock a.m. on the given date. After admission no foods or liquids are given patients who are to have general anesthetic. Those to be operated under local anesthetic are given milk up to eleven o'clock a.m.

Adults over 50 years of age requesting ether anesthesia must be admitted one day prior to operation for a complete physical examination, including blood pressure, blood sugar, etc., and if report is satisfactory operation is done, otherwise, they are referred to the medical clinic for treatment.

No patient is transferred from local to general anesthetic or vice versa without the consent of the tonsil supervisor. General anesthetic is recommended up to the age of twenty years, and to all extremely nervous patients, also to all those who have no control of the gag reflexes.

Before operation a physical examination of skin, nose, throat, ears, heart and lungs is made by an interne. All patients with symptoms of acute coryza, bronchitis (rales), heart murmur, acute catarrhal otitis media, skin eruptions, rectal temperature over 100.4° or any untoward symptoms, are held to be examined by the tonsil supervisor, who decides whether the patient is operable.

Coagulation time is not done as a routine, as it has been found that many patients whose coagulation time is lengthened do not bleed as much as those whose time is normal. Errors may occur when the interne makes the coagulation time test by his breaking the fibrin thread inside the capillary tube; changes in temperature also affect the coagulation time. Heat hastens and cold retards. A few patients who stated that they were bleeders, and whose coagulation time was ten or twelve minutes, were given calcium lactate for from three to six months, or until the coagulation time was lowered to four minutes, this being determined after repeated blood examinations. Though all possible precaution was taken, the operation was followed by severe hemorrhages which necessitated one or more blood transfusions. Recently, electro-coagulation treatment has been used on patients of this type.

The results of all these examinations are recorded on the history card which is pinned on the patient's clothing and accompanies the patient to the operating room. Nurse anesthetists have been employed with excellent results. The first duty of the anesthetist is to compare the name on the card with the name on the adhesive plaster attached to the back of the patient's neck. Anesthetist must then examine the patient's mouth for loose teeth which are extracted before operation begins. Eyes are covered with vaseline wipettes, and castor oil is instilled to pre-

vent ether conjunctivitis. Ether cone method in children under seven years of age, and gas-ether sequence over that age are the methods used. Strict asepsis, as far as possible is maintained, — masks, rubber gloves, and sterile gowns are worn by everyone connected with the operation.

As the patient is wheeled into the operating room the surgeon selects his instruments from a sterile table and places them on a sterile draped Mayo table over the patient. One nurse has charge of the suction tube and another passes the instruments and ligatures to the surgeon. These nurses alternate with each patient.

Members of the house staff operate from twelve-thirty o'clock to two o'clock p.m. daily, each member of the staff having been instructed by the tonsil supervisor until he feels that the interne is competent. During the summer months when the greatest number of patients are operated upon, extra paid operators are employed. The greatest number done in one day was sixty-nine in August, 1934. Not more than ten operations are performed in one hour, and no surgeon operates on more than twenty in one afternoon.

The Flagg laryngoscope is also a part of the equipment of the operating room, and all operators are instructed in its use for asphyxia or collapse.

Adenoids only are removed in children under two years of age. Experience has shown that owing to the small pharynx it is almost impossible to do a perfect tonsillectomy, pieces being left and pillars torn.

We have found that fewer hemorrhages occur if the adenoids are removed first. The adenoid fossa is first examined by passing the index finger up on the right side and down on the left, thus breaking any adhesions from the lateral walls. We discovered more hemorrhages followed the use of the adenoid curette than the La Force Adenatome; the latter is the instrument of choice and is now used exclusively. The first insertion of the adenatome is in the center, then in each lateral fossa. The adenoid fossa is again inspected by palpating with index finger to determine if all adenoid tissue is removed. If any tags remain, the instrument is reinserted until adenoid fossa is clean. The fossa is then massaged with gauze sponge on holder, after which a sponge of dental roll (cut to proper size), with silk thread attached, is inserted into the adenoid fossa and

allowed to remain until the end of the operation. Experience has shown that it is very difficult to palpate with the finger tip to determine whether there is any adenoid tissue left.

Ninety-eight per cent of the adenoid hemorrhages are checked by removal of a piece of adenoid. In a few patients adenoid hemorrhage was controlled by lifting the soft palate and grasping bleeding area with clamp, then applying ligature.

The Sluder and snare method is used in children, snare and dissection in adults. A fresh wire is used in the snare for each patient. The right tonsil is removed either by some modification of the Sluder instrument or snare and dissection method. A dental roll sponge with silk thread attached is inserted into the tonsil fossa. The tonsil is examined for tears in capsule and if found whole the left tonsil is removed in like manner as the right and dental roll sponge inserted. The adenoids and tonsils having been removed, the sponge is taken from the right fossa, and, if necessary, bleeding points are clamped and tied with a ligature. The same procedure is followed on the left side. The sponge is then removed from the adenoid region, and final inspection is made for any further bleeding.

Since making of the ligature always excites comment, a description follows: A tube of No. 2 plain catgut is broken and cut into four equal lengths. At one end a knot is made, the other end being passed from backward forward through loop of knot, and clamp is attached to short end of catgut, leaving a slip noose of the longer end which is passed over the clamp, grasping the bleeding area.

Just before leaving the operating room, the patient is deetherized by inhalation of CO_2, 10%, and O, 90%, and is placed in the proper postoperative position (semi-prone) on the cart, — in bed he is placed in the same position. No patient is sent to the ward with sponge in throat.

The nurse in charge of the operating room records on the history card the time of beginning and end of the operation, the anesthetist, kind of anesthesia used, method of operation, number of ties, pillars torn, teeth extracted, capsule torn, etc.

Whenever an instrument breaks or piece of wire or tooth is missing, an xray is taken from patient's ears to hips before patient recovers from the anesthesia — a report being furnished within fifteen minutes.

All patients operated upon under local anesthetic receive two tablets of luminal grs. 1½, one hour before operation, and are operated upon in a sitting posture. Anesthesia is secured by infiltration of 1% pantocain with gtts. VI adrenalin to one ounce. On returning to the ward the patients are given a hypodermic injection gr. ¼ morphine sulphate, gr. 1/200 atrophine sulphate.

Primary hemorrhages are those occurring in the first twenty-four hours, and which require the service of an interne. Secondary hemorrhages are those occurring after twenty-four hours. Both are controlled by removal of clot, grasping and tying bleed-

HEMORRHAGE FORM

Name.....*Address*..... *Age*..... *Clinic No*.....

CLINIC { Dr. Buckley, Dr. Smith, Dr. Faulkner, Dr. White }

Operator Dr.....Date and time of opt.....

Checked by Dr.....Date of hemorrhage.....

.....A.M. .....P.M.

RIGHT { Upper, Middle, Lower }

LEFT { Upper, Middle, Lower }

METHOD { Clamp, Tie, Plug, Suture, Removal of piece }

ADENOID { Plug, Removal of piece }

PATIENT'S CONDITION { Good, Fair, Poor }

DATE

1st hemorrhage.....

2nd hemorrhage.....

3rd hemorrhage.....

4th hemorrhage.....

5th hemorrhage.....

1st transfusion.....

2nd transfusion.....

ing points, and insertion of plug with gentle but firm pressure. Slight hemorrhages are controlled by application of silver nitrate, grs. 240 to the ounce.

Postoperative hemorrhages are checked carefully as to region, method of stopping, date and number occurring, name of surgeon on service, and the number of sponges left in fossa, also the time when and by whom removed, are all recorded on history card and in the hemorrhage book in each ward.

Severe hemorrhages have been controlled by blood transfusions. As much as 5230 cc. have been given in divided doses. Application of tourniquets to the extremities is sometimes used, but they are not to be left on more than one hour.

The morning following operation all patients are seen by the tonsil supervisor or member of the house staff, who records under postoperative inspection, pillars torn, pieces left, uvula missing; all such defects are checked and initialed in red pencil on the history card. If no defects are found, card is initialed in blue.

All postoperative patients with hemorrhage, cough or temperature over 100.4° remain in the hospital for further observation.

Patients going home are given a postoperative slip calling special attention to the symptoms of cough or hemorrhage. This has enabled us to obtain a fairly good checkup regarding our complications.

MANHATTAN EYE, EAR & THROAT HOSPITAL

Instructions for the Care and Treatment of Patients Following Operation for Removal of Adenoids and Tonsils

PATIENTS MUST BE KEPT IN BED FOR THREE DAYS after operation, then three days in the house. After this, patient may be outdoors at mid-day if weather is fair.

BOWELS MUST MOVE FREELY AT LEAST ONCE DAILY. Give both **ADULTS** and **CHILDREN** milk of magnesia the first thing in the morning.

DIET: Fluids the first 48 hours, ice cream, cornstarch, custard, junket and milk, then well cooked cereal, soups, milk toast and poached egg. **DRINK WATER ABUNDANTLY.**

DO NOT COUGH, HAWK OR CLEAR THROAT AS THIS MAY START BLEEDING.

Parents are requested to bring their children back for examination **one week after operation** on the day written on your clinic card at 1 p.m., except Saturday.

All **ADULT** patients are requested to return as above.

IF ANY BLEEDING FROM THROAT SHOULD OCCUR, RETURN TO THE HOSPITAL AT ONCE.

IF ANY SYMPTOMS OF COUGH DEVELOP WITHIN 10 DAYS AFTER OPERATION, COME TO THE HOSPITAL.

KEEP THIS SLIP AND YOUR CLINIC CARD, and bring the child again in 3 months on the day printed on your clinic card at 1 p.m., except Saturday. The doctor would like to see what benefits have been secured, and whether the condition at that time is satisfactory.

Operations

	Loc.	Gen.	Total	Hem.	Post-Opt.	S.&D.	Sluder	Ties	Operations 5-1-29 1-1-34	Hem. Pri.	Hem. Sec.
1930 NOV-DEC. 1931	1399	10526	11925	474	9246						
1932	1087	10914	12001	579	10320						
1933	1187	9396	10583	548	9394						
Total	3673	30836	34509	1601	28960	10080	24429	55725	52467	3¼%	1¼%

Operations According to Ages — Year 1933

	1-10 Gen.	10-16 Loc.	10-16 Gen.	16-25 Loc.	16-25 Gen.	25-35 Loc.	25-35 Gen.	35-45 Loc.	35-45 Gen.	45-55 Loc.	45-55 Gen.	Pillars T.-M.	Pieces Left
No. opt.	6138	2	1665	103	1151	579	413	358	74	96	14		
Total	6138		1667		1154		992		432		110	1100	194
Hem.	230		99		89		77		37		16		

Hemorrhages

Adenoid hemorrhage	Local	General	Total
115	133	415	548

SYMPOSIUM ON TONSILS

SUMMATION

HARRIS P. MOSHER, M.D.

BOSTON

WHEN I HEARD of this symposium I groaned inwardly. I said to myself, "the same old stuff, treated in the same old way, every discussor talking for his consultants back home." I never was more wrong in my life. Each of the three papers which you have just heard gives something new. Each is short, to the point, and well written. It is one of the best symposia which I have heard for years.

Quite a few years ago Dr. Albert D. Kaiser startled the pediatric world by his publication of a large set of statistics of the school children of Rochester. The result of his figures showed that in his locality the operation of tonsillectomy did not in all cases accomplish what the medical and lay public believed it would. In other words, he found that deep respiratory infections, namely, laryngitis, bronchitis, pneumonia, occur more frequently in tonsillectomized children.

Near the close of his present paper, he states that ten years ago all tonsils were suspected. The only good tonsil was the removed tonsil. His statistics showed that many operated children do not escape infection; in fact, have more infections. The collecting of these statistics was a good piece of work and served a very useful purpose. It led to a reconsideration of the indications for the tonsil operation among those who stop to think before they operate. I answered his statistics at the time by saying that other cities in other parts of the country should assemble similar statistics and get similar results from them before I should be willing to take them at their face value. To prove his case absolutely, all the children should be children living under the same conditions before and after operation. All other variables should be made the same. This, of course, is asking the impossible. Dr. Kaiser has sensed this in his present paper, when he says that the flora of the tonsil varies with geographical localities, and the presence or absence of epidemics, and the race. Another factor, and one which he did not mention, is the mutability of bacteria.

His findings at once bring up the vital question of the relative importance of statistics and of clinical experience. Both may be wrong and neither may be universal.

In his paper he states, in speaking of rheumatic heart disease and nephritis, that it is generally accepted that they have no relation to tonsillar infection. "However," he continues, "unless the teeth, the sinuses, or some other organ is a focus, it is wise to suspect the tonsils. Statistical studies are discouraging, clinical results justify the procedure even if the results are not invariably good." When you are checking up the results of the most frequent of all operations, I almost said of minor surgical operations, which it is not, you need more than a report from one locality. If someone would work out the epidemiology of tonsil infections, and the epidemiology of the results of tonsil operations, if I can use such a phrase, he would be doing a useful piece of work.

Now for other points in the paper. The great point of his paper is his plea for finding an adequate reason before tonsils are removed. He believes with the rest of us that in infancy and early childhood the tonsils have a protective function, although he adds that this cannot be proved, and he prefers not to advise tonsillectomy up to six years of age unless there are repeated attacks of tonsillitis. Dr. Shea, years ago, made me suspicious about removing tonsils in the first five years of life. He, as Dr. Kaiser, thinks the child has more infection instead of less. Dr. Kaiser's indications for tonsillectomy, and his contraindications, are the familiar ones which guide 90% of otolaryngologists.

He thinks the tonsils do not infect the ears, only the adenoid. I cannot agree with this opinion. From my own clinical experience, I do not agree, notwithstanding his statistics, that when the tonsils are removed to prevent recurrent attacks of otitis media, no appreciable difference is seen in the chances of escaping recurrences, except at the ages of one to three, that is, in infants. Adenoids, he says, are chiefly responsible and tonsils have little to do with infecting the ears. I wonder just how he got these statistics. No one in my part of the country takes out tonsils without removing the adenoids at the same time. Occasionally, in infants, the adenoid alone is removed. I understand that in the West, the pediatricians and the obstetricians are doing their own tonsillectomies and mastoidectomies. They certainly catch them young. There is no law against this, but they should remember that they have just as much glass in their dwelling houses as the otolaryngologists have in theirs.

He states that removal of tonsils prevents relatively few children from getting colds. I can understand this, because the common infectious cold is probably the most infectious of all air-borne diseases. Frequent colds, he holds, to be a doubtful indication. I do not agree. I think it may be that adenoidectomy will give greater relief. This half-done operation confuses me, because I do not know any operators who follow this procedure.

When all is said and done, Dr. Kaiser shows that he is human, like the rest of us. When in doubt, he removes the tonsils. Speaking of being human, he states that "systemic infections invariably demand the removal of tonsils at the proper time." Is this humanly possible? I wish he could have been clearer as to just when this time comes and how to recognize it.

In connection with certain diseases, he makes further statements which are helpful. For instance, he states "tonsillar infection has no relation to rheumatic heart disease or nephritis." In patients suffering from rheumatic infection, shown by muscular and facial pain, he advises tonsillectomy. To my mind, these are hazy indications. The reason he gives is that the removal of the tonsils may prevent serious recurrences. This is reasoned out even as you and I would reason it. In an acute rheumatic heart, removal of the tonsils does no good. Acute chorea is made worse by tonsillectomy.

He states, further, that the relation of tonsils to the health and growth of the child is not yet settled. I agree heartily with Dr. Kaiser that good indications should be present before tonsils are removed. I agree that all cases should be thoroughly studied. I agree that some are not. I almost agree with the statement that tonsils are frequently removed without real indications, and that no benefit follows the operation. I object, however, to the word frequently. I am very much obliged to Dr. Kaiser. I am pleased to find that his statistics are worse than his clinical results. I have no patience (this is an interjected remark of my own) with the removal of tonsils in patients from 60-70 years of age, who are really nothing but old wrecks, with the promise of curing their heart disease or arthritis.

I wish that I could show Dr. Nissen's enthusiasm about tonsillar tags as foci of infection in systemic disease. If he is right, I fear for the tonsillar fossa. The average operating skill in tonsillectomies will have to be better than it is at present, with every one who needs the added income feeling that he can do a tonsillectomy anywhere at any time and with any instrument, including the index finger. He is equally enthusiastic about pharyngeal infection as the cause of systemic disease. Here again I prefer to hold my horses a bit. In his original paper, he made the statement that an attack of acute infection renders a tonsil diseased ever afterwards. I am glad to say that he has cut this out. I mean diseased from the practical and clinical standpoint, not from the standpoint of pathology, because from the standpoint of the pathology, every tonsil contains some pathological condition.

Dr. Nissen has done for arthritis what should be done for every disease which is said to arise from infection in the tonsil. I know of no such thorough study ever having been made before. Think of having tabulated the life history of some 96 arthritic patients with reference to tonsil or pharyngeal infection as a primary cause, and a cause of recurrences, and studying exhaustively 400 more! We, as otologists, are very much in his debt for this long piece of work. We are indebted to him also for his clear cut conclusions as to what type of arthritic case to operate, and when to operate.

He said, in conversation, that he believed that in arthritic cases every tonsil should come out. This is pretty strong, but he has studied this type of case more thoroughly than anyone else, and his opinion is to be respected. To speak lightly on a serious matter, this is good news in these depression times. His examination is ideally complete.

Dr. Nissen pleads for a more thorough preoperative study of patients, and a thorough postoperative followup. There are few of us who would not profit by taking this advice seriously. You remember that Dr. Kaiser made very much the same recommendation.

Dr. Jones, in his paper, introduces a specialty within the specialty. He practically has a tonsil clinic apart from the rest of the hospital. He says, very truly, that many operators lead to great variations in the operative results. We all know this. Most of us have not been able to overcome it. Dr. Jones' special service, however, has accomplished it. There is nothing new about his operative technique, but there is about the thoroughness of his preoperative examination, the thoroughness of his records, and his followup — I do not hesitate to say that his special tonsil service is approaching the ideal.

He neglected to say how his clinic is financed. I happen to know that the chief man of this special clinic is paid a salary. The whole thing is on a business basis, and it pays. Again, this is as it should be; it almost runs the hospital. In most hospitals, the administration covertly wishes as many tonsil and adenoid operations performed as the endurance of the staff will permit. In any sizable hospital, the throat department turns into the general fund a sizable amount each year from tonsil operations. This money would, in my opinion, be better employed in paying salaries to establish a special tonsil clinic, following in the footsteps of Dr. Jones.

FURTHER RESEARCH ON EXPERIMENTAL AND CLINICAL SINUSITIS*

OLOF LARSELL, Ph.D.
(By Invitation)

AND

RALPH A. FENTON, M.D.

PORTLAND, ORE.

SINCERE THANKS are due the Research Committee and the Council of the American Academy for its generous continuation of support for these inquiries into the physiology and histopathology of experimental sinus disease and certain clinical applications of previous experimental work.

SYNOPSIS

During 1933-34 our work was directed along several lines, using the cat's frontal sinus in which chronic inflammation closely resembling human types had been produced by inoculation with human hemolytic streptococci. On completion of each experiment the animal was vital-stained by 1% trypan blue and the tissues, fixed in situ in formal-Zenker, were stained by the Maximow technique as reported in all previous research of this series (1930-31-32-33).

1. Young cats of the same litter having unilateral chronic (six weeks') sinusitis were (a) inoculated subcutaneously with 1% peptone; (b) received 1-2 cc. of 1% peptone injected into the sinus cavity by needle puncture; (c) given intramuscular injection of 2 cc. fresh blood from cat's own femoral vein; (d) controls. These procedures, using (a) and (b) peptone as for-

* Research authorized and financed by the Council of the American Academy of Ophthalmology and Otolaryngology, from the Departments of Anatomy and Otolaryngology, University of Oregon Medical School.

eign protein and (c) own blood (autohemotherapy), produced evidences of acute exacerbation, round cell infiltration and perivascular histiocyte accumulation, in these chronic membranes. Peptone subcutaneously seemed to cause the greatest amount of this reparative activity. There was no apparent change from the normal, in the uninfected sinuses. Controls showed no increase of round cell infiltration or histiocytes, plasma cells and polymorphonuclears being most frequent in this edematous membrane of the ordinary chronic type. In a later series, typhoid vaccine was injected into the chronic sinus; there was much epithelial loss, with marked infiltration of the plasma-cell layer of the stroma by numerous polymorphonuclears.

2. Attempts made to demonstrate lymphatic channels of the sinus mucoperiosteum by use of vital staining media were as follows: (a) Normal sinuses: placing liquid India ink outside the exposed upper surface of the sinus lining after chipping away the bony roof; injecting India ink into sinus cavity; (b) normal and infected sinuses: injecting a solidifying 1% agar emulsion of ¼% and of 1% trypan blue into the sinus lumen.

The India ink series failed to show definite black channels in the submucosa or along the bony walls, and caused diffuse blackening of the mucosal surface. Sublingual and deep cervical glands were stained black, but this may have been from the turbinates.

Trypan blue agar ¼% used on animals with the right side infected three weeks, left side normal, membranes secured and fixed after 72 hours, showed much acute edema of the normal membrane, especially toward the ostium. Very great chronic edema and visible capillary dilatation, resembling infected human membranes as removed, were found on the infected side.

A more pronounced surface stain occurred with the 1% trypan blue agar. In two animals, noticeable blue channels, possibly lymphatic, were seen passing forward toward the sinus ostium, both on the "acute" and "chronic" sides, as viewed under the dissecting microscope. These channels were not blood vessels, and apparently were choked with histiocytes and polymorphonuclears. No lymphatic channels were visible by blue stain on the cerebral aspect, roof or floor of the bony sinus cavity or on the corresponding periosteal surface of the submucosa. Time had not apparently elapsed for blue absorption into the deep cervical glands, though the turbinals were deeply stained.

3. Total resection of the right cervical sympathetic was done on cats having bilateral chronic sinusitis. Marked acute edema, congestion and round-cell infiltration took place immediately, in marked contrast to the quiescent, relatively thin membrane full of plasma cells on the side not sympathectomized.

4. Various antiseptic, astringent and stimulant solutions were used in chronic cat sinuses, causing the following changes in membranes which were otherwise similar to chronic human inflammations: (a) merthiolate, 1:1000, severe acute irritation with some recent epithelial loss and polymorphonuclear invasion; there was no apparent exacerbation toward healing; (b) alcohol, 95%, caustic and dehydrating action on superficial layers of epithelium, loss of cilia and marked subepithelial hemorrhage; (c) emulsion of oxyquinoline 1% and scarlet red 1%, causing marked stimulation of reparative effects, with histiocytic and round-cell infiltration, and migration of polymorphonuclears; (d) mercurochrome, 1%, numerous goblet cells, with rather marked epithelial loss, migrating polymorphonuclears and patches of recent round-cell and histiocytic invasion; (e) chloramine, 1:3000, mild acute exacerbation on chronic destructive process; (f) metaphen, 1:5000, fairly mild acute polymorphonuclear and lymphocytic infiltration into severely chronic inflammation; (g) picric acid, ½%, followed immediately by calcium gluconate, ½%, thus precipitating calcium picrate on the membrane, causing very severe acute exacerbation with marked polymorphonuclear infiltration and diapedesis from capillaries.

5. A number of bilateral human cases received a single powerful unilateral therapeutic irradiation by the roentgen ray after methods described last year. Clinical examination two months later disclosed a recurrence of nasal discharge and obstructive symptoms in every case, requiring other methods of treatment. Since a complete tissue report on such cases was made last year, none were operated. It seems evident from this series that irradiation is no guarantee against recurrence, though it seems to promote some degree of fibrosis in simple round-cell infiltration.

6. Six bilateral human cases received diathermy treatments over one antrum twice weekly for one month to six weeks. Four other cases dropped out after a few treatments, claiming relief

after an initial stage of congestion. Of the remaining six, four were much improved clinically, a fifth was relieved of arthritic pain, and the sixth freed from obstructive turbinal hyperplasia, while antral transillumination was improved. Three of the improved cases were operated to secure membranes thus treated. These showed, in addition to the usual marked subepithelial plasma cell layer, a very considerable degree of hyperemia and a good many histiocytes and fibroblasts; evidently a stimulated process of active fibrous repair.

Recapitulation

Foreign Protein: Intramuscular and also sinus cavity injection of foreign protein (peptone, typhoid vaccine and autogenous blood) produces acute exacerbational changes, round-cell and histiocytic pericapillary infiltration, similar to reparative changes aroused by autogenous vaccines.

Lymphatic channels in the sinus submucosa seem to converge toward the normal ostium, and to become more or less clogged during inflammation by histiocytes and polymorphonuclears, thus giving rise to local edema and mechanical obstruction to sinus drainage. These findings require further study by more delicate microscopic and vital staining methods.

Unilateral cervical sympathectomy on cats having bilateral chronic sinusitis brought about active and severe reparative congestion, edema and round-cell infiltration, while the other side remained quiescent, with predominant plasma-cell infiltration and no attempt toward repair.

Several powerful liquid antiseptic solutions occasionally used as irrigations of the healing operated antrum and other wounds (1:1000 merthiolate, 1:3000 chloramin, picric acid ½% followed immediately by calcium gluconate ½%, and 95% alcohol) were found to produce further losses of epithelium, polymorphonuclear invasion, and apparently to delay the healing process. Mercurochrome 1%, metaphen 1:5000, and oxyquinoline sulphate ½% emulsified with ½% scarlet red, seemed to promote round-cell and histiocyte infiltration to a limited extent, without, however, improving the repair tendencies of the mucosal surface.

Our opinion expressed last year, that the therapeutic use of the roentgen ray may produce some fibrosis following round-cell destruction, but that no healing of suppurative changes in the

submucosa can thus be made to take place, was borne out by exacerbations of varying severity on every one of ten irradiated cases called back for examination and treatment two months after therapeutic irradiation.

Diathermy applied for one month's treatment twice weekly to one side in numerous bilateral maxillary sinusitis cases produced rapid clinical amelioration in nearly all; operative removal of tissues disclosed much recent hyperemia with many histiocytes and some fibrous repair, evidently stimulated by the local and enforced temperature elevation.

Topics requiring further and detailed study, with eventual clinical application of results obtained, include: lymphatic obstruction to sinus drainage during infection; local reparative effects of systemic agents acting upon the sympathetic (acetylcholin, pilocarpin, adrenalin, ephedrin); attempts to break up or metamorphose plasma-cell infiltration through physical or biochemical agencies.

Details of Experimental Studies

Experimental work was carried out with the technical assistance of Dr. Lyle M. Bain and Mr. Wallace Shearer. Clinic cases were managed by Dr. Victor Adix, Resident in Otolaryngology. Roentgenologic treatments were given under the direction of Dr. Ivan M. Woolley, and diathermy was administered by Dr. Arthur Jones.

Use of Foreign Protein — Young cats from the same litter received right-sided frontal sinus inoculation with human upper respiratory hemolytic streptococci, and after six weeks, a time established in past experiments as sufficient to produce changes simulating those of chronic human sinusitis, the following substances were administered:

Cat 501 — 2 cc. of 1% peptone injected into infected right sinus cavity. Seventy-two hours later, 3 subcutaneous injections of trypan blue 1% for vital staining; animal killed, routine fixation of upper half of skull in Zenker-formalin and dissection in toto as in previous experiments. The right membrane was slightly thicker than the left, and quite adherent to the bony wall. Microscopically, cilia were retained, there was marked subepithelial edema with numerous plasma cells, but numerous peri-

capillary round cells and histiocytes were found, indicating an acute exacerbation as a result of the peptone injection. The left membrane (uninfected) was normal except for an unusual number of goblet cells; there were more histiocytes than normal about small blood vessels.

CAT 502 — Subcutaneous injection, 2 cc. 1% peptone, otherwise same procedures; findings practically the same as 501.

CAT 503 — 2 cc. of cat's whole blood withdrawn from femoral vein and immediately reinjected intramuscularly. Gross findings same as 501 and 502; infected sinus rather small, dissection difficult. Microscopically, epithelium contained many goblet cells, occasional drifts of plasma cells in areas of marked edema, in which were a few histiocytes about small vessels. Left membrane normal. This cat showed less marked, but evident, reparative effects as a result of autohemotherapy.

CAT 504 — Same procedures as 503. Slightly more marked round-cell and histiocyte pericapillary accumulations on the infected side; no polymorphonuclears, apparent increase in size of serous glands.

CAT 505 — Control, from same litter, showed only the usual plasma-cell accumulation in an edematous membrane, with no evidence of recent congestion or histiocyte accumulation on the infected or normal side.

Study of Lymphatics — CATS 506, 507, AND 508 — Both frontals exposed by carefully chipping off bony roof, exposing membrane; on the right side, membrane was opened, on the left it was kept intact. Five cc. of India ink diluted with normal saline solution was placed on each membrane and the external skin and pericranium sutured. Membranes fixed in situ after 72 hours. No definite channels shown either on bone or toward ostia, but ink deposits, possibly from blackened turbinates were found under the tongue and in superior cervical glands.

In a later series, an emulsion of agar 1% with trypan blue, and with radioöpaque oil (Parke-Davis) was injected into the sinus cavities of animals infected on the right side three weeks before, left side normal. This emulsion was permitted to act for 72 hours, producing an acute inflammation of the normal side and allowing gradual infiltration of the vital staining agents into the epithelium and tissue spaces, after which the usual fixation and dissection were done. Cat 520, in which radioöpaque oil was used, had no right frontal, and the injections reached the right orbit. Slight thickening of the left membrane was noted, without any definite lymph channels.

CAT 518 — Trypan blue agar ¼%, and CAT 519, trypan blue agar 1%, both showed much acute edema of the uninfected membrane, especially forward toward the ostium. A more marked surface stain occurred with the 1% emulsion. Very great chronic edema and capillary dilation were visible on the infected side; these membranes grossly resembled edematous human tissues. Both animals showed a few distinct streaks of blue passing forward near the ostium; these were not blood vessels, and were apparently choked with histiocytes and polymorphonuclears. The bony walls of the cavities showed no blue streaking, and the surface of the membrane facing the bone disclosed no streaks or channels on the upper, posterior or lower surfaces. Edema was most marked toward the ostium on both the acute (uninfected) and chronic (infected) sides. There was dark turbinate staining but no gross evidence of blue dye in deep glands, which were not studied microscopically.

Effects of Sympathectomy—CATS 510 AND 511—Both frontals infected six weeks; right cervical sympathetic sectioned, tissues secured 72 hours later and destruction of sympathetic proved by dissection, below superior cervical ganglion. Grossly, right membranes were thickened and edematous, with blood vessels injected; left membranes very slight thickening, apparently healing after injury. Microscopically, on the sympathectomized side lymphocytes, plasma cells and polymorphonuclears were scattered throughout, with beginnings of cyst formation in several places; a rather severe chronic infection with recent mild exacerbation and acute edema and thickening of the membrane. On the opposite side the membrane was but slightly thicker than normal, with evidences of healing of the previous infection.

Results of Local Application of Antiseptic Solutions — CAT 512 — Bilateral streptococcus viridans infection for eight weeks; the right sinus injected with oxyquinoline sulphate 1% and scarlet red 1%, aqueous emulsion, tissues recovered and fixed after 72 hours. The membrane was thick and much injected. Microscopically, the mucosa was greatly thickened, filled with lymphocytes, plasma cells and some histiocytes. The epithelium was much injured, and destroyed in patches. Polymorphonuclears were numerous near the surface, some migrating through the epithelium; the sinus cavity contained masses of them. The tunica propria was very edematous and congested, containing numerous plasma cells. The total picture was that of a chronic, infected membrane with an acute exacerbation, the latter apparently due to the oxyquinoline-scarlet red.

The left sinus of this cat was filled with 1% aqueous mercurochrome solution. The entire membrane presented the usual gross thickening of chronic infection. Microscopically, part of the membrane gave the picture of mild chronic inflammation, with

thickened mucosa filled with plasma cells, lymphocytes and some polymorphonuclears. The epithelium over this area was thickened, with numerous goblet cells and intact cilia. On the opposite side of the sinus, however, the epithelium had been destroyed, leaving only basal cells, and no goblet cells or cilia. In patches the epithelium was gone. The tunica propria of this part of the sinus showed acute reparative inflammation with numerous polymorphonuclears passing through the remnant of epithelium; some histiocytes, and numerous plasma cells, were present.

Cat 513 — Infected bilaterally for six weeks with hemolytic streptococci, was injected in the right sinus with 95% alcohol. Tissues were secured and fixed in situ after 72 hours. Grossly, the membrane was very much thickened and reddened. Microscopically, the usual picture of chronic infection was present, with numerous plasma cells, etc. The epithelium was injured throughout, with cilia largely destroyed; in some parts only the basal layer of cells remained. Elsewhere the epithelium was entirely absent, and beneath these regions there was marked hemorrhage. The total picture was that of a chronic membrane which had been severely inflamed by the recent use of alcohol locally; there was no indication of recent infectious activity or of any stimulation toward repair.

The left sinus of this cat was injected with aqueous solution of merthiolate 1:1000. The sinus membrane was greatly thickened. Microscopically, the mucosa was extremely thickened, filled as on the right side with polymorphonuclears, plasma cells, etc. The epithelium was almost totally destroyed, that remaining filled with migrating leukocytes. The tunica propria was thicker than any other of the series, containing numerous plasma cells, macrophages, and near the epithelial surface many polymorphonuclears. Some fibrosis was evident in the deeper part of the tunica propria. The total picture was that of a chronic, severely infected sinus membrane upon which had been superimposed a very severe and destructive inflammatory flareup, apparently caused by the merthiolate solution.

Cat 514 — Bilateral streptococcus infection for six weeks; right side, no sinus found on dissection, the 1:5000 metaphen used having entered the orbit. The left sinus, injected with 1:3000 aqueous chloramine solution, showed the usual picture of chronic infection, with a mild acute exacerbation from the chemical used.

Cat 515 — Nine weeks infected bilaterally with streptococcus viridans, was injected on the right side with 1:5000 aqueous metaphen solution. This cat, and Nos. 516 and 517, showed more marked response to the original infection than did most of the previous animals reported on in this series. Notably thickened

on removal, the microscopic sections of the membrane showed a mild chronic type infection, with a recent exacerbation indicated by numerous polymorphonuclears. The epithelium was destroyed in patches.

The left frontal, injected with merthiolate 1:1000 aqueous solution, showed marked gross thickening. Microscopically, the stroma was very thick, with numerous plasma cells throughout. Near the surface were many polymorphonuclears, of which considerable numbers were migrating through the destroyed epithelium. All blood vessels were greatly dilated, with some extravasation. As in Cat 513, merthiolate 1:1000 apparently produced definite irritation and destruction.

Cat 516 — Similarly infected, was found to have no right frontal, which made the injection of ¼% silver nitrate solution act merely as a vital staining medium for the right orbital tissues. The left sinus, filled with typhoid vaccine, showed the usual gross and microscopic changes due to chronic infection. The epithelium was much injured, with leukocytic migration going on. Outer layers of the tunica propria were crowded with polymorphonuclears. This showed a definite acute reparative exacerbation on top of the old process.

Cat 517 — Similarly infected, received an injection of picric acid ½%, followed through the same needle by calcium gluconate ½%, simulating the picric acid-calcium carbonate treatment of osteomyelitis wounds advocated by Stewart. The membrane, greatly thickened, showed microscopically severe epithelial damage with patches of denudation. Numerous plasma cells and some polymorphonuclears were found subepithelially. Leukocytic migration was frequent, and the lumen was filled with polymorphonuclears. The tunica propria was greatly thickened and edematous. The total picture is that of an old, chronic membrane with recent exacerbation, in some places very severe.

The left frontal was filled with 1% peptone solution. The recovered membrane was relatively thin. Histologically, the epithelium was tall, with numerous goblet cells. The cilia were partly destroyed, and some migration of leukocytes was taking place through the epithelium. The tunica propria was thin, but contained some plasma cells. Some residual infection was evident, but healing had gone further than in others of the series at nine weeks. The peptone did not cause a flareup, but appears rather to have helped the healing process. This membrane has the nearest approach to normal appearances of any of the experimentally infected sinuses.

Results of Diathermy in Human Membranes — Three out of six bilateral cases of maxillary sinusitis treated from eight to eleven times over the left sinus only by diathermy were operated

four to five weeks after the end of the course of treatments. Grossly, the membranes in one case were still much thickened, and more hyperemic than the average polypoid case; in another, that irradiated had become definitely thin and good transillumination had returned before operation. The untreated membranes presented the usual picture of masses of plasma cells, cysts, and marked edema. After diathermy, even in the thick membranes, and in spite of marked subepithelial plasma cell infiltration, a considerable number of histiocytes could be located near the capillaries, which were distinctly dilated. In the thin diathermized membrane, there were numerous histiocytes and fibroblasts, with fairly marked congestion without edema, and a number of eosinophiles. Evidently the continuing hyperemia from this form of heat therapy assists in bringing in cells which are concerned with repair by fibrosis.

RESEARCH STUDIES IN VISUALIZATION OF THE VASCULAR SUPPLY OF THE HEAD AND NECK

O. Jason Dixon, M.D.*

KANSAS CITY, MO.

One of the unsolved and very important problems in otology is the diagnosis and anatomical location of a thrombus within the sigmoid sinus. Since Zaufal,[1] in 1880, first described and operated successfully for the removal of this very destructive lesion, no accurate means has as yet been devised for a positive diagnosis without opening into the sinus.

On account of the infected adjacent bone, the opening of a normal sinus is always a distinct menace to the life of the patient. The problem of diagnosis becomes particularly difficult when there is bilateral mastoid infection of equal duration and severity, which so frequently occurs in children.

Considerable thought and much work has been given to this problem, but up until now the only approach to its solution has been the work of Toby and Ayer, who thought that by compressing the internal jugular in the neck on the non-involved side, and that by increasing the cerebro-spinal fluid pressure, that they were able to secure additional positive evidence for the location of the site of the lesion. This obviously presents many disadvantages, and the information obtained is necessarily of uncertain value.

Drainage of the cerebro-spinal fluid, in the presence of a localized dural infection, is also thought sometimes to play a part in the intracranial dissemination of the infection. This is rather a major procedure to perform upon a very sick patient. Neither does direct inspection of the exposed sigmoid sinus, at the time of operation, give accurate and positive information of

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1. Zaufal, quoted by Ballance, C. A., *Surgery of Temporal Bone*, II, p. 360.

the pathological findings within the vessel. The outstanding clinical signs and symptoms may also be confused with other pyogenic lesions, which are far removed from the mastoid process.

In an attempt more accurately to diagnose the presence of a thrombus within the sigmoid sinus, I have hit upon a method,

FIG. 1 — (Dixon) — Xray of bilateral viable muscle implant in the external jugular — 30 days postoperative — re-canalization — Sodium iodide 50% — 5 cc. injected into each sublingual vein.

more or less by accident, while carrying on some research problems in the study of vascular lesions in the dog.[2] In order not to disturb the process by surgical removal, and more clearly to

2. *Experimental Studies in Vascular Repair*, Journal A.M.A., October, 1934 Issue, Volume 103, pp. 1129-1134.

determine and to demonstrate the vascular changes which were occurring after the insertion of viable muscle implants into the external jugular vein of the dog, I decided to use radioöpaque

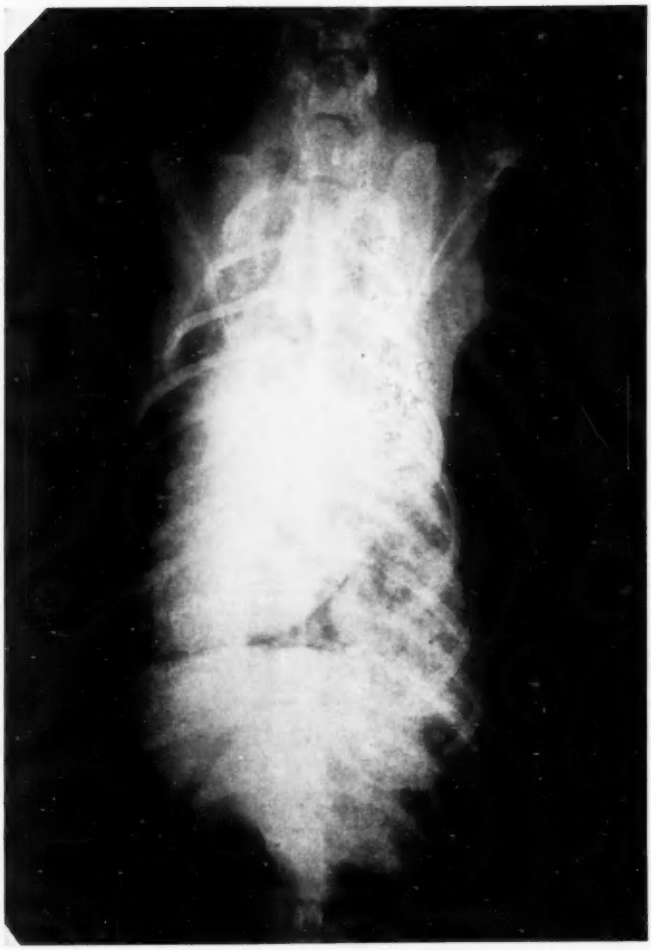


FIG. 2 — (Dixon) — Xray of dog immediately after death which had had sublingual injection of 5 cc. of 50% Sodium iodide, showing the marked pulmonary changes and the retention of sodium iodide in the lung.

media, which I injected into the sublingual vein. As sodium iodide has for some time been used, both as a diagnostic measure for radiological studies as well as for intravenous therapy, I first started with this opaque media.

The picture which I obtained by the injection of small amounts was very striking (Fig. 1), and clearly demonstrated the lesion which I was attempting to show. Quite to my surprise, however, and much to my chagrin, all of the animals which received this injection died within twelve to twenty-four hours

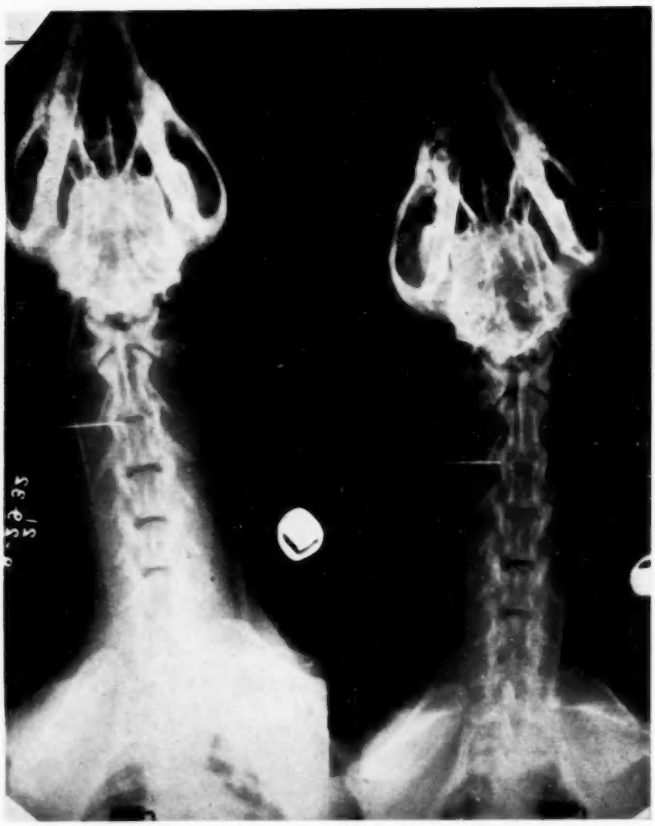


FIG. 3 — (Dixon) — Xray showing the very faint shadow of the internal jugular vein with sublingual injection of plain Neo-skiodan solution which has not been concentrated or chilled (without the addition of glucose). No details can be made out and the collateral veins cast a very indistinct shadow.

afterwards. (From 2 to 5 cc. of solution consisting of 50% sodium iodide and 5% starch was given, which was well within the human lethal dose). I have learned by further investigations that similar accidents occasionally happened when sodium iodide was given to the human. The pathological findings at

necropsy revealed that intravenous sodium iodide was extremely irritating to the vascular endothelium. All of the dogs presented a highly inflamed and irritative destruction of the vascular endothelium of the lung. Where they lived long enough, that is, eighteen to twenty-four hours, the entire lung became water-

FIG. 4 — (Dixon). FIG. 5 — (Dixon).

Xray showing sublingual injection of concentrated Neo-skiodan and glucose chilled to 55° F. and injected into the sublingual vein of the dog, showing the collateral compensatory venous circulation with the fine tributaries clearly shown.

logged with this inflammatory exudate, and in some of the cases there was an acute pleurisy with extensive effusion (Fig. 2). The kidneys also showed this same vascular change, and I was quite obviously obliged to turn to another source in order to ob-

tain this desired roentgenological effect without the sacrifice of my experimental animals.

About this time a new opaque media called "Neo-Skiodan" had been found to be an absolutely inert product, which held the iodine as an inert chemical until it was completely and quickly thrown off by the kidneys. Unfortunately, however, the injection of this media in the liquid form was too dilute to give a good picture. The channel of the internal jugular vein could be followed (Fig. 3), but no vascular details could be made out, and in no way did it give as satisfactory a picture as that produced by the sodium iodide.

By concentrating the Neo-skiodan and then saturating with glucose, a much better picture was obtained (Fig. 4) and quite by accident I learned that if this solution were chilled prior to its injection, that it did not become so rapidly diffused with the flowing venous stream, and cast a much better shadow. In fact, this shadow is so clear-cut and distinct that the collateral vein can be plainly shown (Fig. 5) through the entire skull of the dog's head. It is a well-known law of physics that two fluids do not mix until they reach an equal temperature.

Additional valuable information has been obtained by the injection in the saphenous vein for the study of artificial lesions which had been produced in the vena cava, as for example, one of my research studies was the closing of the incision in the vena cava by the application of the raw surface of a strip of anterior rectus muscle taken from the dog's abdomen, which had been left attached at one end in order that it would carry its own blood supply and remain viable.

These xray studies, which were confirmed by necropsy specimens, as well as physiological evidence, conclusively proved that the vena cava was not completely obstructed by the formation of the thrombus at the site of the artificial lesion. The xray evidence did confirm, however, the microscopical and anatomical findings that only a mural non-obstructive thrombus was formed. Even when large dogs, with a marked amount of abdominal fat were used, and only 6 cc. of a 55% solution of Neo-skiodan was injected slowly into the small saphenous vein of the dog near the foot upon one side, the outline of the vena cava was distinctly shown, and the shadow was not obscured by crossing over the spinal column. In addition to the localization of the mural thrombus, the point of entrance of the renal veins could be de-

FIG. 6 — (Dixon) — Xray of inferior vena cava in the dog following the injection of 5 cc. of concentrated Neo-skiodan and glucose into the right femoral vein. The dark oval-shaped shadow just below the rib is the point where the viable muscle was applied to the artificially-produced defect in the vena cava.

termined, as this localized and increased venous bloodstream, diluted the opaque blood within the vena cava at the point of entrance and diminished the shadow at this point (Fig. 6).

In none of the animals, where skiodan was used repeatedly, was there any systemic reaction. Necropsy studies failed to reveal any evidence of any vascular irritation whatever, except that after the second injection the sublingual vein, which was quite small, often became obliterated, which I believe was due to the local distension and the trauma produced by the needle.

Now the problem which confronts us in the use of this chemically inert and extremely satisfactory media for the determination of vascular lesions in the human is the method of inserting the solution distal to the site of the venous disturbance. Naturally, we are dependent upon the venous stream to carry the solution. Except within the skull, this presents no particular difficulty, but at once we are confronted with a difficult problem, if we wish to make xray studies of the sigmoid sinus. It has certainly been well proven that any injury to the intima of the sigmoid sinus is apt to be followed by the formation of a thrombus which may become infected. We have long since discarded the dangerous method of inserting a needle through the exposed sigmoid sinus for the aspiration of blood. However, in certain selected cases, where all other means have failed in the differential diagnosis of the involved sigmoid, the injection of this opaque media above the site of the suspected lesion, is worthy of trial, and is much less serious than opening directly into the sigmoid sinus to determine the location and extension of a thrombus, if present. In some cases the emissary vein could be used, and, by going well up toward the torcular end, the point of injury should be kept well away from the site of infection.

I recently saw in consultation a nine-year-old boy, who had had, four days previously, a bilateral mastoidectomy with exposure of both sigmoid sinuses. One sinus was covered with old granulations and showed a rather extensive involvement of the vein wall. The other was smooth and appeared normal. As the child had a septic temperature, a positive blood culture, and was developing a metastatic abscess, it was quite obvious that he had a septic thrombus in one of the sigmoid sinuses. To our surprise we found by direct injection of Neo-skiodan by a small needle into the exposed sinus that the normal-appearing sinus

was the one that was thrombosed. I have seen this Neo-skiodan solution accidentally enter the subdural space and cause no disturbance to the patient whatever. The chief point in the technique of securing good xray studies is to take the picture immediately while the solution is entering the bloodstream.

This method will, I believe, also be distinctly advantageous in the diagnosis of bulbar and internal jugular vein thrombosis. It might also be used in certain select cases for the differential diagnosis of cavernous sinus thrombosis, by injection into the angular vein. This, however, is fraught with serious hazards, and one should always appreciate the importance of not injur-

FIG. 7 — (Dixon) — Thrombosed sigmoid filled with opaque media showing the definite point of obstruction by the shadow which was cast following the injection of opaque media into the exposed sigmoid. The needle at the instant that the xray was made was not in the sigmoid sinus.

ing the vein in the presence of infection.[3] The brain is such a large vascular organ that it is difficult to isolate distinct changes by intravascular shadows. Hemangiomas, however, and other vascular lesions, do present the possibility of a more complete study by this method. It may also be used as a diagnostic measure for hemangiomas of the tongue, the pterygoid and pharyngeal plexus, and in other areas about the head and neck. In the

3. O. Jason Dixon, *A Departure in the Management of Infections of the Sigmoid Sinus*, Archives Otolaryng., II:453-57, 1925 (Nov.).

dog, I have attempted to produce shadows within the cranial venous sinuses by injecting this opaque media directly into the common carotid artery, which carries it to the brain, where it is returned by the venous system (Fig. 11A). Comparatively speaking, the dog has a very small brain, and, therefore, requires much less blood than the human.

FIG. 8 — (Dixon) — Xray of right mastoid, D. R., Mercy Hospital, showing the subdural extravasation of skiodan and glucose that was accidentally injected into the subdural space. Borders of the infected sigmoid sinus can also be made out. Patient made an uneventful recovery. (See Fig. 9). This accident shows the necessity of using a non-irritating and harmless fluid.

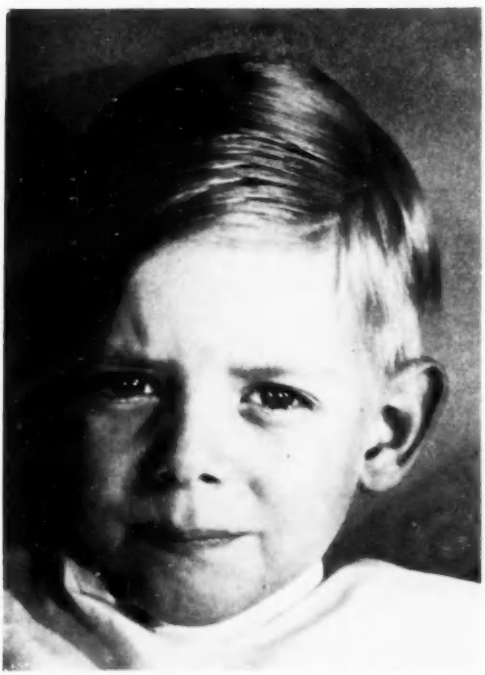


FIG. 9 — (Dixon) — Photo of patient, D. R., Mercy Hospital, three months after the injection of opaque media into the exposed sigmoid sinus which accidentally entered the subdural space. (See Fig. 8).

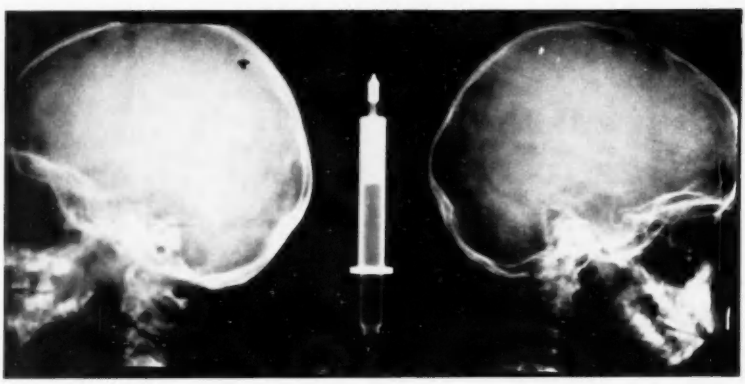
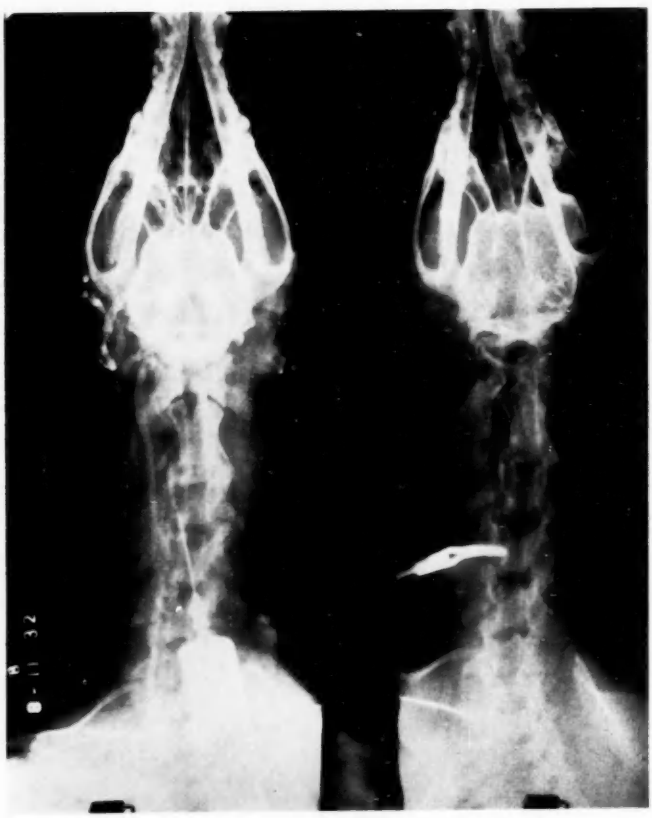


FIG. 10 — (Dixon) — Lateral xray of skull taken prior to injection of opaque media directly into the sigmoid sinus. This patient, S. F., Mercy Hospital, had had a viable muscle in the sigmoid sinus 29 months before. Xray (*B*) shows the vertical shadow of the opaque media in the sigmoid sinus which apparently has re-canalized. Xray of hypodermic syringe shows relative opacity of the solution.

The maximum amount of blood in the dog's head goes to the muscles of mastication and to the salivary glands, as it is by the use of the latter that he maintains his thermal control. I have been able, however, to show, by radiological evidence, vascular shadows in the external ear (Fig. 11B), but these, I am inclined to believe, are in the arterial supply. Also, we are confronted with an additional major procedure if we attempt to inject this opaque media into the carotid artery of the human.



A B

FIG. 11 — (Dixon) — Injection of contrast media directly into right common carotid artery. Venous return through right internal jugular vein plainly shown. Right external jugular vein removed by previous operation. Shadow is plainly seen down to point of obstruction. Apparently the large rapidly-moving arterial stream dilutes the contrast media so quickly that shadow only shows faintly near the point of injection. Xray (*B*) shows artery clamp on right common carotid after exposure and injection of contrast media three minutes showing injection. Media had already been so dissipated that only faint shadow is remaining along course of left internal jugular vein.

SUMMARY

Xray studies for suspected lesions within the sigmoid sinus, jugular bulb and internal jugular vein may be accurately and completely studied by the injection of opaque media distal to the site of the lesion. This opaque media is inert and harmless to the patient. To increase the shadow, it should be given in a concentrated solution of glucose. The picture must be taken just prior to the administration of the last cubic centimeter, that is, while it is flowing into the vein. It may also be used as additional evidence of intra-abdominal vascular lesions, or to show any alterations of the normal contour of the vena cava. The solution should be cooled to a thickness that will just permit its continued flow through the needle, 55° to 65° F. These vascular studies are by no means limited to the areas previously described, but are of particular value in differentiating which sigmoid sinus is involved. It may also be used for a confirmatory evidence of any suspected venous lesion, such as arteriovenous aneurism, or thrombophlebitis in any area.

* * * * *

I am deeply indebted to Dr. R. M. Isenberger, Professor of Pharmacology, University of Kansas, for valuable coöperation and in whose laboratory all of these experiments were conducted. I am also deeply obligated to Mr. Mark Carroll, my technical assistant, for many valuable suggestions. The xray studies were made in the laboratory of Dr. Galen Tice of the Department of Radiology, University of Kansas, and I wish to express my appreciation of his untiring assistance.

NOTE: Since this paper was read, seventeen injections of various concentrations of opaque media have been injected into the intra-cranial sinuses, with no unfavorable disturbance to the patient. These include injections into the exposed longitudinal sinus, as well as the sigmoid sinus.

Various concentrations of the opaque media were used, and a more detailed description of the technique, as well as the different solutions, will be presented in a separate paper.

DISCUSSION

Dr. Samuel J. Kopetzky, New York: The paper that Dr. Dixon presented resolves itself into a search for the answer to two questions, with one of which we as clinicians are very much concerned, with the other we may eventually become concerned when we will be able to translate the findings from the laboratory back to the bedside.

I know of no problem which gives the clinical otologist more concern than a bilateral otitic infection, signs and symptoms of sepsis, and the question of which side is involved, or whether both are involved. Dependent upon the answer to that question, in many instances, the results are proportionate.

Often, when that clinical problem presents itself, valuable time is lost, while various methods are called to our aid to make the determination, and all of the methods that we have at present at our disposition are not totally adequate. The best that we have had until now has been the Toby-Ayer estimation, and in many instances that is equivocal. The more recent modification of that test by the observation of the eye grounds while one or the other jugular vein was compressed, is likewise equivocal. Dixon comes along with a definite, positive means, as yet in its very preliminary stages, but he is on the road to a solution of one of our most perplexing problems, and I am glad, indeed, to stand here and applaud and congratulate this Academy for the opportunity of hearing that which, at the present time, might only be considered as a preliminary report, but which from the experimental work on the animal and from the few cases which Dixon has already submitted to his test, have shown such promising results.

It is true at the present time he must expose the lateral sinus and puncture it. We have been exposing the lateral sinus and puncturing it to draw blood, to determine whether or not it was patent or thrombosed, and that in itself, that test, is inadequate for very, very many reasons for which there is not time at this particular moment to discuss.

The other problem that Dixon presents, namely, the re-canalization of a sinus after the implantation of living muscle, is a problem which, from my clinical standpoint, is the most important part of what he presented here today. That is academic at the present time, and no discusser has a right to express his own individual opinion unless he has also done the work or observed the experiments. In regard to that aspect of Dr. Dixon's presentation, I will say that I will await his further work and his further reports.

I think that this type of paper, not sterile research, not academic research, but research with the clinical bent that it has, is fruitful in bringing us something which eventually will help us in our work.

Dr. Frank R. Spencer, Boulder, Colo.: When I heard Doctor Dixon present this subject before the Triological Society in Atlantic City two years ago last spring, I was a bit skeptical about the possibility of retrograde venous currents in the large intracranial venous sinuses. However, these veins are without valves in man and this will permit a retrograde current.

Dr. Andrew A. Eggston, a pathologist at the Manhattan Eye, Ear and Throat Hospital in New York, presented a paper before the last meeting of the American Otological Society last April in which he said, "In a series of eight cases of cavernous sinus thrombosis, two were of otitic origin as a result of retrograde extension through the petrosal veins to the cavernous sinus." He furthermore said, "These are of great interest, because the petrosal veins are efferent vessels of the cavernous sinus and the infection traveled against the circulation." His article has just been published in the September number of the Annals of Otology, Rhinology and Laryngology, page 672.

This retrograde current is also a factor in producing either a brain abscess or a meningitis, as particles of infected blood clot are loosened from the septic thrombus.

A study of comparative anatomy shows that there are no valves in the veins from the head and neck in man, and valves are present in the dog. The absence of valves should, therefore, make it easier for man to have these infections from retrograde currents than the dog, with which Doctor Dixon has done so much of his work.

Dr. O. Jason Dixon, closing: I wish to thank Dr. Kopetzky and Dr. Spencer for their very kind remarks.

I meant to suggest the use of a piece of viable muscle, not necessarily viable muscle, to place over the opening which is made in the sigmoid in case it is not desirous to go ahead and open it, if it is not thrombosed. I believe that has advantages over gauze, which I haven't time here to show.

I was very happy to note that Dr. Spencer mentioned the retrograde involvement. I forgot to stress that. As you recall some of the slides, the vein was much smaller toward the head than it was toward the heart, where thrombosis had originally occurred.

We are up against a very different problem in the dog when it comes to infections. I tried out a very large series, trying to infect the thrombus which blocking produces, using such virulent organisms as streptococci from patients who had just died of otitic meningitis. The dog wags his tail and goes merrily on, even when 10 cc. are injected into the vein. However, if a little of the nasal discharge from a dog suffering from distemper is injected, an entirely different situation occurs.

I haven't had the time nor the inclination to find out what the organism is which causes distemper in the dog, because that is quite a different

problem, but he does react to his own organism, and he doesn't react the same to ours.

I think this will probably have more use in the diagnosis of lesions such as the lower extremity, the liver, and the inferior vena cava and, possibly, with better improvement of technique, in the intracranial vascular lesions aside from the particular one which we are now discussing.

I had in mind the expectation of injecting this medium into the angular vein in suspected thrombosis of the cavernous sinus. I don't believe it will do any particular harm, although I don't think it should be used in the presence of a violent infection in that area, but it may serve to make a differential diagnosis very beautifully, I believe, in the cavernous sinus thrombosis where we occasionally are confused with orbital abscess and other lesions about the eye.

SYMPOSIUM:

THE CONSERVATIVE TREATMENT OF THE NOSE, THROAT AND EAR

THE NOSE AND THE ACCESSORY SINUSES

W. E. Grove, M.D.

MILWAUKEE

The purpose of my discussion of this subject is to outline the principles of conservative treatment of the nose and sinuses. It is not my idea to create any argument between the proponents of conservative and radical surgery, both of which have a very definite place in nasal therapeutics.

The abnormal, pathological conditions of the nose and sinuses which call for any sort of treatment can, with very few exceptions, be placed in four general groups; malformations of the structural framework of the nose; infections; allergic conditions; and neoplasms. Only the treatment of infections and of allergic conditions will logically fall within the scope of this discussion, because the treatment for malformations of the nose and for neoplasms is purely surgical and frequently radically so.

In considering the treatment of infections of the nose and sinuses, one must consider, first of all, the defense mechanism of the organism and then how that defense mechanism can be assisted and stimulated by treatment.

Referring to the nose and accessory sinuses, the first line of defense against infection consists in the ciliary activity of the nasal and sinus mucosa and the mucinous blanket layer which overlies the ciliated epithelium.[1,2,3] To these must be added, though of minor importance, the action of air currents within the nose during inspiration and expiration, and the variations of air pressure within the sinuses during respiration.

The second line of defense consists of the phagocytic action of macrophages and microphages; the action of opsonins, lysins, and agglutinins and the reactions of natural immunity, both general and local.[4 5]

Of the many influences which tend to undermine the natural resistance of the mucosa the most important are: diet, physiological effects, allergy, and trauma. By physiological effects we refer to chilling, ventilation, chemical irritation, metabolic fluctuations and obstruction to drainage. Linton[5] regards all of these factors as important because any one of them may be the deciding factor in causing a break in the defense of the mucosa. Schmidt[6] has called attention to the action of cold on the skin and mucosa of the upper respiratory tract in causing a prolonged contraction of the blood vessels of the skin and mucosa. Because of this prolonged vasoconstriction, the defense forces can no longer be easily mobilized and the ever-present bacteria find a favorable field for development.

Our treatment of infection of the nose and sinuses, whether conservative or radical, must be designed to assist the natural defenses of the mucosa and restore its function to normal. The normally functioning mucosa of the upper respiratory tract with its mucinous layer, its ciliary action and its reticulo-endothelial elements is marvelously efficient in protecting the individual against infection.

Under ordinary circumstances the offending bacteria are engulfed in the mucinous blanket and are propelled, with varying degrees of rapidity, toward the pharynx, esophagus and stomach, where they are destroyed. When, because of obstructed drainage or physiological effects, such as persistent chilling, bad ventilation, chemical irritation and metabolic disturbances, the action of the ciliated mucosa is interfered with, mass action of the invading organism takes place, the epithelium is penetrated, the sinuses are invaded. Leucocytic exudation to combat the invading organism results in free secretion or pus in the nose and sinus cavities to such an extent that stagnation occurs, the movement of the mucinous blanket is still further hindered and a vicious circle initiated. Our defense mechanism has broken down.

Our efforts at this point must be concentrated upon the reduction of the swollen mucosa, the removal of stagnant secretions and the stimulation of the mucosa to resume its normal activity. This can be accomplished by applications and packs of

various kinds to shrink the sinus ostia, by irrigations of various sinuses to remove stagnant secretions and by other efforts to increase the proper ventilation of the sinuses.

In this connection, the experiments of Lierle and Moore[7] are illuminating. These investigators experimented upon the action of various drugs on the ciliary activity of the mucosa in man, dogs and guinea pigs, and came to the following conclusions:

1. Tap water and distilled water, when applied to the mucosa of the upper respiratory tract, causes a slowing of the ciliary beat.

2. Three per cent ephedrin hydrochloride is not detrimental to ciliary activity, but at times slightly increases it.

3. Five per cent cocaine hydrochloride is not detrimental to ciliary activity, but 10% and 20% solutions produce definite slowing with good recovery.

4. Mild silver protein solutions of 5%, 10% and 20% produce an initial speeding of ciliary activity with a subsequent slowing, probably due to the water solvent.

5. One-half per cent eucalyptol is not detrimental.

6. One-half per cent and, to a greater degree, 1% menthol has a mildly depressing effect.

7. One per cent thymol, ½% and 1% eucalyptol are definitely detrimental to ciliary activity in the order named.

8. A 1-1000 epinephrine hydrochloride, a 2% zinc sulphate and a 2% mercurochrome solution, in the order named, are definitely detrimental to ciliary activity.

9. One-half per cent silver nitrate solution is immediately and fatally detrimental to ciliary activity and in no instance was it possible again to start ciliary beating after its application.

With the above facts in mind we will probably make some changes in our applications of medication to the nasal and sinus mucosa. We can increase natural drainage and stimulate ciliary activity by the application of some of the above mentioned drugs to the nasal mucosa and more particularly to the sinus ostia. Medication, more particularly ephedrin solution, can be directly introduced into the sinus cavities by the displacement method as advocated by Proetz.[8] Whether the displacement method is any additional help in diagnosis seems to be a mooted question, but Proetz,[8] Frazee[9] and others have rather definitely shown

that it is of much benefit in conservative treatment of nasal and sinus infections.

When our efforts to assist the mucosa are blocked by stagnation within the sinuses, the stagnant secretions can be removed by normal salt solution irrigations. Such irrigations can be carried out on all the sinuses except those of the ethmoid capsule. In many cases the normal ostia can be catheterized. Puncture of the antrum has been performed since the very beginnings of rhinology. Watson-Williams[10] has popularized the puncture of the sphenoid, and Van Alyea[11] has recently advocated the puncture of the floor of the frontal sinus where the normal opening could not be probed.

Drainage and ventilation of the antrum can be still further improved by a window resection of the inferior meatal wall; that of the frontal sinus by infraction of the middle turbinate or the removal of the operculum, and that of the sphenoid by removal of its anterior wall.

When the above outlined measures have failed, when the sinus infection has passed from an acute or subacute into a chronic condition, when the lining mucosa has been replaced by a pyogenic membrane with enormous increase in the connective tissue elements, then, and then only, has our conservative treatment failed to benefit the patient and more radical measures must be resorted to.

Diet, climate and sunshine play a large rôle in the prevention and cure of sinus infections. The fact that colds are practically unknown in the arctic, relatively rare in the semi-arid belt, and very common in regions where wide variations of temperature and humidity prevail deserves consideration from the standpoint both of prophylaxis and treatment. Shea[12] says, "The older my sinus patient happens to be, the quicker I send him searching for the sun." Certain it is that sinus patients who cannot find comfort in the climates of rapid temperature and moisture changes are much benefited by living in regions like southern Florida, southern California and on the eastern slopes of the Rockies. If sunshine is unavailable, Shea recommends daily ultra-violet radiation to the entire body and within the nose. I heartily concur in the first half of this recommendation because the action of ultra-violet radiation, particularly from the sun itself, is a very potent factor in assisting the body to auto-immunization against respiratory infections. I have, however,

never seen any benefit from the direct application of ultra-violet therapy to the nasal mucosa itself.

With respect to diet, Jarvis[13] believes that the absence of fat in the diet of young individuals tends to increase lymphoid tissue in the throat and that the lack of uncooked vegetables in individuals from 20 to 30 will increase the catarrhal discharges from nasal and sinus membranes. Cody,[14] working on the effects of vitamin deficiency diets, maintains that vitamin A in the diet is necessary to maintain the nutrition of the nasal, aural and tracheal mucosa. Prophylactically, vitamin A seems to increase the resistance to upper respiratory infections in children. Therapeutically, its chief value is to improve the nutrition of the nasal and aural mucous membranes in acute and chronic infections.

According to Cody, a diet, deficient in vitamin B, is identified with a nasal syndrome consisting, essentially, of a postnasal discharge and the posterior tips of the middle turbinates are smooth, white, moist and slightly thickened.

Vaccines — The normal sera or plasma have some anti-bacterial activity (Harkness[4]). When infection takes place auto-immunization begins, and the cells of the reticulo-endothelial system begin to throw out antibodies to combat the bacterial antigens (Fenton and Larsell[15]), in addition to increasing enormously the numbers of microphages and macrophages. Clinically, we can definitely assist this auto-immunization process by diet, hydrotherapy, heliotherapy and vaccines, both specific and nonspecific.

The best results from specific vaccine therapy are obtained from autogenous vaccines used over a considerable time. However, for more immediate action some form of nonspecific protein therapy may be employed. For this purpose we can use parenteral injections of such substances as lactogen, boiled milk, typhoid vaccines and omnadin (Shea[12] and Mithoefer[16]). In my own hands, omnadin has been very effective.

In the prevention of ordinary seasonal colds, I have found very successful the use of certain stock vaccines, worked up gradually to an immunizing dose, which dose is repeated once or twice monthly during the winter months.

Before leaving the subject of vaccines, I must call attention to the subject of local immunity as produced by the specific non-

filterable antivirus solutions as worked out by Besredka.[17] Besredka and his co-workers have shown, both in vitro and in vivo, that bacteria cannot live and develop in the presence of their own homologous antiviruses, either in the skin, in the eye, or in the peritoneal cavity. Kolmer,[18] in cases of chronic bronchitis and bronchiectasis, made intrabronchial injections of specific antiviruses, and he believes that, in addition to the bacteriostatic and bactericidal action for its own specific organism, the antivirus owes its therapeutic properties to stimulation of both microphagic (polymorphonuclear) and macrophagic (reticulo-endothelial) phagocytosis, together with a local antibody production. Mulso,[19] using a stock antivirus, had marked success in rapidly healing the mouth lesions of agranulocytosis.

If all these facts be true, it would appear profitable to inquire into the possible therapeutic benefit to be derived from intrasinus applications of specific antiviruses.

Allergy — A constantly increasing number of nasal and sinus conditions which formerly masqueraded under the designations of vasomotor rhinitis, hyperesthetic rhinitis, recurring colds, polyposis, and the like, have more recently been recognized as manifestations of allergic states and conditions. Efficient treatment depends absolutely upon correct diagnosis and this diagnosis must identify the offending allergen or allergens. In arriving at this diagnosis a most careful and painstaking history is of prime importance, as Mullin,[20] Hansel,[21] Rowe[22] and others have pointed out. This history must be a general inquiry into the patient's present and past illnesses, habits and idiosyncrasies. It must also include a careful inquiry into the medical history of his immediate ancestors. This history is then supplemented by physical examination, by cytological examinations of nasal smears, by differential blood counts and by skin sensitization scratch tests. It has, however, been pointed out by various workers in this field that skin tests are frequently negative in cases where true allergy exists. Therefore, too much reliance should not be placed on these tests alone. The elimination diets of Vaughan[23] are frequently helpful in making a diagnosis.

The diagnosis once made, the treatment will entail the close coöperation of rhinologist, internist and allergist. Any operative treatment of pure allergic manifestations in nose and sinuses must be scrupulously avoided. Bearing in mind, however, the

frequent coëxistence of allergy and infection in the same individual (Mullin,[20] Hansel,[21] Menkin[24]) the treatment of allergic condition must include the treatment of the infection, be that in the sinuses, teeth, tonsils or any other part of the body.

Knowing the rapid and extreme swelling of the allergic mucosa, with its tendency to polyposis, great care will be exercised in nasal surgery with this exception, that, where allergy and sinus infection coëxist, the same rules as to the care and treatment of this sinus infection will obtain as in the nonallergic individual.

Before closing, mention might be made of the value of free iodine and calcium therapy (Feldmann[25]) as an adjunct in the treatment of allergic states.

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SYMPOSIUM:

THE CONSERVATIVE TREATMENT OF THE NOSE, THROAT AND EAR

THE PHARYNX

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OBSERVING CLINICIANS, since medical history is recorded, have considered the mouth and pharynx "the mirror of the body." Clinical medicine and research are constantly corroborating the observation that most pharyngeal pathology is secondary to disease in the nose and sinuses, the lower respiratory tract or to some general systemic disturbance.

Pharyngeal diseases from a consideration of management and treatment may be classified as:

1. Primary infections implanted upon the pharyngeal mucosa by air- or food-borne organisms.
2. Pathology due to continued infection in the nose or lower respiratory tract or in the lymphoid or glandular structures in the pharynx itself.
3. Non-infectious irritants.
4. Pharyngeal manifestations of general alterations or diseases.
5. New growths.

The naso-pharynx, the oro-pharynx, and the hypo-pharynx must, to some extent, be considered as distinct entities, since they differ in anatomy and function.

NASO-PHARYNX

The naso-pharynx is essentially a part of the nose, being lined by the same ciliated, mucus-covered membrane, which must always be respected. It contains structures of distinct importance, such as the pharyngeal end of the eustachian tube and the adenoid. Its function is to convey air, supplement the local defense mechanism of the nose proper, permit aeration and drainage of the ear cavities, and, by its musculature, provide a barrier between the nose and pharynx proper. The wall of the naso-pharynx, being placed at almost right angles to the nose, is subjected to maximum trauma from air currents and irritating substances in the inspired air. Because of natural architecture, nasal discharge is also directed into the naso-pharynx, frequently with continued infection or irritation. The adenoid tissue, usually the first part of the pharyngeal lymphoid ring to become an active offender from chronic infection or hypertrophy, may continue infection, or obstruction, or, by adherence of its lobes, provide the necessary cavity for a Tornwaldt's abscess.

The above recital of architecture and responsibilities of the naso-pharynx is made to emphasize the importance of an area which too often is grossly and radically treated by the application of toxic, irritating drugs, or permanently damaged by careless removal of adenoid tissue.

The oro-pharynx, highly vascularized and enervated, is assigned the double duty of transmitting air and food. It is subjected to infections and trauma by way of the mouth, as well as from the nose and naso-pharynx. It is lined by a non-ciliated squamous type epithelium, which permits of certain therapeutic liberties not applicable to the naso-pharynx. The oro-pharynx contains the faucial tonsils, which are usually the second part of the lymphoid ring to lose their usefulness in a breakdown of pharyngeal lymphoid defense. It also contains innumerable lymph nodes and glands, which may become hypertrophied or infected.

The hypo-pharynx assumes the highly-specialized function of diverting food into the squamous-cell-lined esophagus and air into the trachea, with its columnar ciliated epithelial lining. The structures of greatest importance in this region, from the standpoint of treatment, are the larynx and the tonsillar portion of the lymphoid ring.

Primary Pharyngeal Infections

Acute pharyngeal infections, resulting from an overpowering of the local and general defense by a virulent organism, whether it be a so-called cold infection, influenza, a milk-borne streptococcus, or one of the acute exanthemata, is largely a matter of intensive general treatment. The extreme vascularity of the pharyngeal walls favors rapid absorption of toxic material, and the great number of mucous glands and lymphoid tissue in the pharynx, if involved, tends to increase and continue the toxemia. Complete rest with proper elimination and supportive treatment is of major importance. Local treatment, while of secondary importance, can assist in limiting the infection, and add comfort to the patient. Local treatment is of value both from the point of bacterial destruction and symptomatic relief.

The naso-pharynx is usually the first portion involved in air-borne infections and is least amenable to local treatment in the acute stage, since the same anatomical defense mechanism must be conserved as in the nose. The application of toxic or irritating drugs to this area in acute infections with their resulting reaction, depresses the local defense and may easily extend the infection into the eustachian tubes and middle ear.

Local treatment of acute infections in the oro-pharynx is more conducive to good results, with less likelihood of interfering with the local defense mechanism. It has been the custom in the past, however, to use drugs of a caustic nature as bactericides, such as strong silver solutions, iodine, etc. This practice can not be supported either theoretically or in practice in the light of our present knowledge of tissue defense. The use of non-corrosive bactericides, if used frequently as hot gargles, is probably beneficial. These, however, must be used at frequent intervals to provide the bactericidal effect as continuously as possible, and doubtless the heat is as effective as the bactericide. The use of weak phenol solutions of non-coagulating dilution is antiseptic and adds much to the patient's comfort by their local anesthetic effects. As a local application, all the known antiseptics have been used, which, in itself, is an admission that none are entirely satisfactory. One of the most effective agents for application to an acutely inflamed oro-pharynx is a five to ten per cent solution of a guaiacol in gylcerine. In addition to being an effective antiseptic, guaiacol produces a pleasing anesthetic effect in the throat for a considerable length of time, although

its immediate effect is a distinct burning sensation. When applied to a mucous membrane, this mixture is retained for a prolonged period to continue its bactericidal effect.

Acute laryngeal infections also must be treated with due consideration of the existing anatomy. The tendency for œdema to occur in the laryngeal mucosa, following acute pyogenic infections, makes attempts at its prevention of first importance. This can best be done by absolute voice rest, the use of drugs for mild, general and tracheal sedation, the avoidance of irritating drugs in the larynx, and the use of massive cold applications over the larynx. Marked œdema of the larynx from pyogenic infection is not infrequent in children, often to the point of dangerous asphyxia. When dangerous asphyxia is imminent, the logical procedure is low tracheotomy to prevent general exhaustion and fluid accumulation in the lung spaces. Intubation or bronchoscopy in these cases produces a high percentage of laryngeal strictures.

Chronic Pharyngeal Pathology, such as chronic infective pharyngitis, pharyngitis sicca, and chronic laryngitis are purely symptoms, the various causes of which must be ascertained and eliminated, if possible. Probably the most frequent cause is chronic nasal and sinus disease, with recurrent or continued infections in the numerous mucous glands and lymph follicles in the pharyngeal wall. Local treatment indicated in these conditions is mild stimulation of the mucous glands; this is best accomplished in the pharynx by silver nitrate in two to five per cent solutions. In chronic laryngitis, the instillation of three per cent creosote in olive oil is particularly effective. Occasionally, the mucous glands and small lymph follicles of the pharynx become chronically infected, hypertrophy takes place, and these remain as continued sources of irritation after the nasal or other primary infection is removed. Surprisingly few of these remain, however, if all nasal or other factors are eliminated, so that conservatism calls for repeated search for etiologic factors. If these cannot be found, destruction of the infected glands or lymph nodes is necessary by surgical removal, careful cauterization with chemicals, or by electro-coagulation.

Chronic Lymphoid Infection

Pharyngeal pathology, due to infection in the lymphoid structures in the pharynx, is a subject of major importance in this

discussion, as it is concerned with the tonsil and adenoid problem. Acute infections in the tonsils and adenoids are essentially a condition requiring general treatment, with local attention, as described for an acute infectious pharyngitis. It is not a conservative or logical act to traumatize an acutely or chronically infected mass of lymphoid tissue by attempts at curettage or suction of the tonsil crypts. If a distinct abscess is present, free incision is indicated for drainage.

Xray irradiation of some chronic pharyngeal diseases has an important place in therapy. Its value in the treatment of infected lymphoid structures is to reduce the amount of lymphoid tissue and facilitate drainage from unclosed crypts. It is valuable as an aid in the occasional case where surgery may be contraindicated, but it does not entirely eliminate infection. Irradiation is particularly valuable in chronic pharyngeal infections, such as carriers of diphtheria, Vincent's or actinomycosis.

Regarding the logical or conservative attitude toward the removal of lymphoid tissue from the pharynx, in spite of the fact that the adenoids and tonsils are apparently not true lymph glands, there is evidence that when in a reasonably healthy condition they act as part of the first line of defense against the common infections. There can be no logical argument in favor of tonsil and adenoid removal, except that they have not only lost their defensive power, but actually harbor infection, which is being disseminated, or they are sufficiently hypertrophied to produce obstruction. Hypertrophy of the tonsil and adenoid may be either the result of infection or of a systemic glandular or chemical dyscrasia, which should be determined and corrected. When evidence accumulates that these structures have been overwhelmed, as complete a surgical removal as possible is the method of conservatism.

The adenoid is the earliest and most serious offender in younger children, and its removal, without permanent damage to the naso-pharynx, is a difficult procedure. Conservation of the basal portion of the pharyngeal mucosa, underlying the adenoid tissue, is essential to prevent scar formation and an irritable naso-pharynx in the future.

Conservative treatment of chronically diseased tonsils calls for complete surgical extirpation as against electro surgery, because of its higher percentage of complete removals and satisfactory results.

Specific infectious diseases, common to the pharynx and larynx, are tuberculosis, syphilis, Vincent's infections, and in certain sections, actinomycosis.

Tuberculous infections in the pharyngeal wall, or in the larynx, are best treated locally by electro surgery, either by removal of granuloma or surface cautery on the walls of the pharynx, or multiple stab puncture in the larynx. The varied therapeutic agents, such as chaulmoogra oil, acids, heliotherapy, etc., used locally in these infections, have not proved of sufficient value to consider their use as conservative.

Syphilitic lesions of the larynx are only local manifestations of a general disease, and must be treated in a general way.

Vincent's infections in the pharynx can, in the majority of instances, be eliminated by frequent application of sodium perborate to the infected area. If, however, the infection is severe or inaccessible, one should resort at once to moderate doses of arsphenamine intravenously. The local application of this drug is of exceedingly doubtful value.

Non-infectious irritants of any nature, the most common being tobacco smoke, are a source of irritation to the pharynx in certain individuals. This can be demonstrated by observing an irritable pharynx with a cough subside after smoking is eliminated. Elimination of smoke as an irritant is a valuable, but little used, therapeutic procedure in pharyngeal diseases. The addition of cooling drugs to cigarettes, in the light of recent investigations of the effect of menthol on the respiratory mucosa, indicates further irritation from these.

We must not overlook the fact that many of the chronic pharynges, especially of the irritable congestive type, often with great quantities of mucous secretion, is merely a superficial reaction of an altered general chemistry, or the result of a vascular disease, where circulatory power is impaired. The much discussed mucous membrane changes in the nose, due to altered general processes, has an older counterpart in the so-called rheumatic or gouty throat of the past, and is deserving of constant thought in attempting to diagnose and treat pharyngeal diseases.

Malignancies of the Pharynx

Recent developments in the treatment of malignancies in the pharynx, based upon the pathological picture present, have

added much to conservatism and offer greater possibilities of a cure. Every pharyngeal growth observed demands immediate biopsy, as early diagnosis is the essence of conservatism. Irradiation of a malignancy, for limitation before biopsy or any surgical procedure, would be ideal. This is not practical as a routine, but should be practiced in all highly suspicious cases. The present-day classifications of carcinoma cannot be accepted as a positive indication of radio-sensitivity, but, generally speaking, the epithelial growths classified as type one and two cell construction are not radio-sensitive, and conservatively demand wide surgical removal with irradiation following for its limited effect. Carcinomas classified as type three and four are apt to be more radio-sensitive. To this latter group, also, may be added sarcoma and lympo epithelioma. These tumors are best treated by prolonged fractional irradiation to a saturation point, combining both xray and radium. If, however, massive growths are present, their removal facilitates irradiation. Conservative management of laryngeal malignancies calls for the same type of prolonged fractional irradiation as the pharyngeal malignancies, but is demanding, more than ever, early open operation for complete surgical removal, rather than by endoscopy.

SYMPOSIUM:

THE CONSERVATIVE TREATMENT OF THE NOSE, THROAT AND EAR

THE EAR

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MEDICAL AND surgical treatment in otolaryngology would seem to have been in a peculiarly transitional state for the last decade. Several important factors have contributed to this.

FIRST, *the growing realization that the influence of nutrition and biochemistry upon all upper respiratory pathology has not only been underrated, but too often ignored.* There is sufficient evidence to believe that research investigation in this field already well advanced may change the entire perspective of both routine and surgical measures in the not too distant future, and that adequately regulated body chemistry will supplant much that is being imperfectly done at the treatment table — the time-worn analogy of reaching the trouble at its source.

SECOND, *the almost finality which has now been attained in the technique of radical surgery.* Perhaps sufficient time has now elapsed to balance the ledger and estimate experiences in end results. This has automatically occurred in large degree, as one could almost forecast the popular trend in radical mastoid and sinus surgery.

THIRD, *the better understanding of allergic reaction and its intimate relation to medical and surgical decision.* Probably only the threshold of this important correlation has yet been passed, and its breadth of application is not yet sure. The intranasal and sinus birthright to allergic manifestation should claim priority in this symposium discussion.

FOURTH, *the confusing advent of manifold newer antiseptic preparations* and the rapid supplanting of time-honored therapeusis now experimentally proved inefficient. It would seem that medical as well as surgical treatment in recent years has reached a radical stage.

The honest specialist is confronted with such queries as this: Are routine nasal irrigations, oropharyngeal applications, tubal treatment and massage overdone? This and other symposiums on the conservative handling of the ear, nose and throat are a response to the transitional unrest cited above, and should further stimulate biochemic research.

It must not be interpreted from the foregoing that the intention of this paper is to champion the nonsurgical handling of ear pathology. One must be in perfect agreement with Shambaugh,[1] who summed up his discussion of clinical problems in otitis media by saying that no form of local treatment can ever be expected to cure a running ear where an indication exists for radical surgical measures.

It is the purpose here to discuss borderline cases and argue where possible for the conservative plan. The symposium title was made "conservative" rather than "nonsurgical" to broaden this discussion. No effort will be made to review the voluminous literature on the subject, which all may read. This will be simply a chronicle of clinical observation and experience.

The three most important conditions in the ear to be considered from the conservative standpoint are infection of the external canal, acute and chronic middle ear involvement, and mastoid infection. Labyrinthine infection, tubal stenosis, progressive deafness might indeed be added, for there is wide variation in routine, but the latter conditions are not so germane to the title.

The greatest safeguard for conservatism rests in the preliminary history and record of each patient. Nothing but emergency surgery, and in the case of the ear seldom that, should prevent a careful analysis of the patient's own and family history, habits of life, response to variations in climate, dietary régime, elimination, associated medical conditions, depressive influences and idiosyncrasies. To this should be added minute laboratory study, blood and urine studies, including the daily von Schilling shift of immature neutrophiles, and basal metabolism. Ear tests, tuning fork and audiometer should be taken when feasible, regardless of known deafness, and a preliminary caloric test for

vestibular tract may give you surprising leads. Voluntary nystagmus, gross neurologic reactions and the nasopharyngoscopic picture of the pharyngeal opening of the eustachian tube should be noted. In even doubtful cases mastoid roentgenograms, eye grounds and fields, and a cautionary blood culture should be taken. Allergic reactions even in a study of the ears are appropriate. One cannot visit the offices of some of our finest otologists without being impressed with their care and precision in collection of almost voluminous detail and their masterly methods of analysis.

This careful preliminary study in even simple ear conditions is important for the following good reasons: 1st, valuable data are on record for comparison in later ear study and treatment; 2nd, intracranial mischief is often discovered from routine study even in the semi-quiescent stage; 3rd, information is often derived which shows that this patient does not really belong in otologic hands and the proper reference can be made; 4th, the ear with its tinnitus, vertigo and hallucination hearing, very frequently has a psychic bearing. The patient's confidence sequent to the realization of a cautious study is well worth having, for optimism is therapeutic in itself. One grave caution is the question of finance. Laboratory work does cost money, but the otologist should have sufficient influence to obtain this for patients of limited means. 5th, Otolaryngologists wish to share their achievements for the benefit of science. Results of study and to the realization of a cautious study is well worth having. For experience are worth but little unless fortified by accurate and trustworthy records. 6th, As a final reason for this somewhat lengthy preamble to conservative treatment, specialists in every field may be roughly divided into two groups — one with radical, one with conservative bent. If one has the operative habit, his subconscious reaction is operative. This is at least effective, but not always protective to the patient. If he is at heart conservative or wishes to become conservative, he will emphasize this preliminary investigation and be less liable to interpret basic conditions by superficial irregularities.

Infections in the External Canal

Three conditions might be pictured in discussion here. A. Furunculosis of the canal; B. disorders of the epithelium; C. disturbances arising from the presence of exostoses or other malformations.

A. *Furunculosis of the canal:* This is too often not a simple matter and tries both physician and patient. The long and deep incision to the periosteum in the early stages of this disorder is usually neither wise nor conservative. Here, as elsewhere, nature needs a period of time to wall off and protect surrounding tissues. While direct transmission to middle ear, mastoid or meninges is perhaps unlikely, there is no evidence to prove that surgery will greatly shorten the old-time antiseptic or poulticing methods, persisted in to absolute pointing and well-nigh automatic elimination. This is not in the same category as the infection of the upper lip and angle of the nasal alæ, but conservatism invites culture, vaccine and systemic treatment to build up furuncular resistance.

B. *Disorders of the epithelium:* There is some consensus of feeling, that infections involving the lining membranes of the ear canal should be put under dermatologic supervision as the lesions can be so manifold. It should always be borne in mind how sensitive is the external ear canal — how easily vascular blebs are formed in topical manipulation. It would be conservative to avoid unnecessary paracentesis of the tympanic membrane because of failure to diagnose myringitis bullosa and herpes of the drum head, canal mycosis or rupture of abscess through fissure of Santorini, as pointed out by Hunter.[2]

C. *Exostosis and other malformations:* Exostosis deep in the canal may cause much concern and the surgical temptation is great. Fortunate is the surgeon who makes approach through postauricular route rather than through the canal, if he must operate. Usually conditions present are complicated by the abnormal anatomy rather than directly caused by it — careful diagnosis and treatment will more often obviate the necessity of interference.

Acute Otitis Media

The two most popular subjects in any otolaryngologic session are removal of the faucial tonsils and incision (paracentesis) of the tympanic membrane. The tympanic problem revolves about the question as to when the membrane should be opened. There are many variations in acute intratympanic involvement from the simple injection, varying degrees of myringitis, serous fluid level with or without air bubbles to the massive bulging with toxæmia and febrile reaction where there is no debate.

With the evidence of definite mastoiditis, delay in abortive measures would be unjustifiable. Many cases of mastoiditis doubtless occur from ultraconservative delay, and some doubtless occur without obvious middle ear disturbance. Nevertheless, countless middle ears are opened unnecessarily — often resulting in chronic suppurative otitis media. Many argue that a middle ear with catarrhal serous fluid, not drained, contributes to middle ear deafness. Careful study of many ears in our office with tuning fork and audiogram check-ups would, however, tend to disprove this.

In doubtful ears, with neither mastoid nor systemic threat, disregard the simple hyperæmia and treat as a myringitis. Packard once pointed out, in a ward review, that the short process and malleus line in clear relief tends to negate post-tympanic fluid. Why not immediately attack the pharyngeal end of the tube — ephedrine, cocaine or other measures to obtain tubal drainage, and await results? Even if tympanic opening be necessary, repeated incisions are of doubtful value as the middle ear becomes filled with scleroses and granulations.

Chronic Suppurative Otitis Media

It is impossible to avoid linking this with the question of the radical mastoid operation — so often employed for its relief. The study of chronic suppurative involvement of the middle ear requires some review of nature's basic efforts in relief, and one becomes intimately involved in discussion of Aschoff's and Wittmaack's theories of infantile otitis media and sequence of the diploetic and sclerotic mastoid states, which in principle prevent the occurrence of a coalescent mastoid involvement, and which have been so admirably presented by Almour.[3] In infantile otitis media neanatorum, due to early respiratory forcing of meconium and other substances into the middle ear, antrum and mastoid become filled with hyperplastic connective tissue — and pneumatization is halted with a resultant sclerotic mastoid; in the true infantile suppurative otitis media, due to bacterial invasion from the eustachian tube or through blood or lymph from an infected external canal, fibrosis occurs with marked contraction of the subepithelial embryonal connective tissue and again a halt of pneumatization occurs with a resultant diploetic mastoid. Both of these conditions, with various interesting defense mechanisms in the different types of tympanic perforation, change the middle

ear suppuration to a somewhat local problem and naturally invite the multitudinous methods and medicaments presented for its control. From a conservative standpoint this situation places the radical mastoid in a position of last resort. Chronic suppurative otitis media may be chronic, recurrent or intermittent, but this does not greatly change the principles of treatment.

From the conservative position and assuming that in every middle ear invasion there is at least initial mastoid involvement, antrum or anterior lateral cells, the first thing to do is to determine by accurate xray and other diagnostic measures the sclerotic, diploetic or other state of the mastoid. The second point is to determine the defense mechanism in tympanic membrane. A central large type perforation should tend toward self-limitation when proliferating mucosal membrane comes into conjunction with marginal squamous-celled activity in the perforation wall. This type, according to the work of Asherson,[4] alone responds favorably to the zinc ionization treatment which might shorten nature's more tedious repair.

The third step in conservative procedure would be the effort to obtain nonsurgically the results of radical method, which would customarily be either ossiculectomy, the radical or modified radical mastoidectomy, intratympanic mastoid antrum approach, as in the operation of Tobey,[5] and perhaps, by your permission, some such modified attic drainage as that advocated by the writer. This effort should be twofold, to produce drainage and apply antisepsis. In suppurative involvement of the middle ear, the cavity has become filled with adhesions, scleroses, organized granulations and fibrosis until the initial drainage has become a narrow, tortuous and irregular canal. Remembering that destruction of tissue and retained fluid will tend to increase the discharge, an effort should be made to clear away cholesteatoma, crusts and other débris, by peroxide, alcoholic, boric and glycerinated solutions, and then make sure that the drainage exit is clear. Dilating the existing exit in safe areas such as the posterior quadrant can do no harm. New membranes will form rapidly once the infective element is removed. A cardinal point is the use of antiseptic agents — no amount of antiseptic treatment per the auricular canal route will be of any avail unless it actually reaches the intratympanic infection, and therein lies the value of actual irrigation by attic canula. Iodine powder or other agent must pass through the tympanic opening to produce

result — xray radiation, ultraviolet and infrared rays, high frequency currents, mercury quartz light and similar methods may prove the exception to this rule but their results have not yet been sufficiently attested in experienced hands to fully prove their efficacy. Many antiseptic preparations have been used, of which the most popular has been the iodine powder advocated by Ledermann.[7] Others have prepared similar powders in their own laboratories. Hurd[8] suggests surgical diathermy to remove adhesions and infected lymphoid follicles around mouth of eustachian tube, Nelson[9] ephedrine solution directly in middle ear, Hetrick[10] postural drainage, Fox[11] dental drying rolls, Kirkland[12] chromic acid, Watson-Williams[13] magnesium sulphate crystals, Shaheen[14] and others subcutaneous proteid injections. Di-Sulpharmin,[15] Picratal,[16] oxyquinolin tartrate,[17] meroxyl and acetic acid[18] have all been the subject of favorable experience reports. All these procedures carry but one suggestion — cases are individual. Treatment, both systemic and local, will depend for success largely upon the patience and skill of the otologist rather than by any particular medication.

Mastoid Infection

In mastoiditis, immediate operation is seldom required. There is usually sufficient time to study the patient and fortify his resistance. Many a mastoid is aborted by prompt and adequate care of the middle ear, and there is no evidence to indicate that when the patient has escaped operation by such treatment the ultimate function of the ear has been injured. An acute mastoid in experienced hands is given an opportunity for walling off during a period which might vary from one or two days to even weeks. The danger of rapid intracranial invasion from too hasty operation, in the presence of subacute meningeal symptoms of toxic character, will thus be avoided. Such a delay is trying for surgeon, patient and his friends and often induces a change of otologist. Experience has demonstrated that ultimate confidence is gained from a firm stand.

In this transitional period, with the prospect of great awakening from scientific research now well in hand, it behooves clever otolaryngologists to stand in the front rank, grasp proven guide posts and at least present the conservative rather than the surgical trend of mind.

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SYMPOSIUM:

THE CONSERVATIVE TREATMENT OF THE NOSE, THROAT AND EAR

SUMMATION

SAMUEL IGLAUER, M.D.

CINCINNATI

IT IS A VERY difficult thing to discuss three papers at once. It reminds me very much of a baseball game where the manager says to a pinch-hitter, "Go up and knock a home run and bring those three fellows in." These distinguished members who have spoken to you have covered the subject very completely, and I shall not detain you very long.

Of course, we must define our terms, and the question is what is conservative and what is radical. This is very difficult nowadays. In politics, what one man considers conservative, another considers Bolshevistic. But, generally speaking, I suppose that conservatism, on the one hand, means nonsurgical treatment, and radicalism means the use of surgical procedures. However, you cannot draw a fast line, because sometimes what appears to be radical surgical treatment is really conservative for the life and health of the patient.

There is no doubt that with the perfection of our instrumentarium, especially in the nose and throat field — when we go to any convention we find there are more instruments for doing nose, throat and ear operations than for all the rest of surgery put together, — having purchased one of these instruments, we have a natural itch to use it.

If you will analyze what has been said this afternoon, I think you will find that most of the gentlemen are pointing out the natural defenses of the body against infection.

When I was a student the professors talked about "*Vis medicatrix naturae*." That was just a keen observation on their part that Nature does cure.

At this convention we have had two interesting demonstrations, for instance, one by Dr. Proetz on "The photography of the ciliated epithelium," and one by Dr. Taylor on "The effects of diving and swimming in lowering the resistance of the body."

Dr. Proetz showed conclusively that if one chilled the mucous membrane after it had been removed, or even in the living rabbit, that the cilia

slowed down. Most of you have seen Hilding's remarkable pictures in which he demonstrates that it is not so much the cilia as the mucus, the mucous blanket, I believe he calls it, which covers the cilia, which rolls up the bacteria and prevents their entrance into the mucosa.

It is important, of course, in considering infection and immunity, to remember that the epithelium of the body, not only in the mucous cavities but also on the skin, protects us from invading bacteria, and anything that we do in the nose, which interferes with the epithelium, promotes or invites the entrance of bacteria.

There is one other thing about the ciliated action in the nose. It is very hard to explain from a rational point of view why the cilia in the nose should carry the secretion into the nasopharynx, but that is the thing, I think, we have inherited from our very remote ancestors, because in some of the amphibia and in the immature human embryo, we find ciliated epithelium in the esophagus. Of course, if we were made over, we would reverse the action of the ciliated epithelium and push everything out of the nose instead of into the pharynx.

In the same way, most of the ostia of our nasal sinuses are wrongly placed for an animal that walks on its hind feet. We have heard this afternoon, too, that many of our pet remedies are not what they seem to be. For instance, menthol has been condemned, and that will be very hard on the Vick Company. We also find that nitrate of silver, which is a very excellent thing in the eye, is not so good in the nose. Well, probably the difference is that in the eye you do not have ciliated epithelium.

We have also heard about the mobilization of microphages and the macrophages at the point of irritation. This has been studied in various parts of the body, not only in the antrum. This is very important, that, when an irritation occurs, Nature sends an army of two types of cells, monocytes and the macrocytes, to fight off the infection, and we are learning more and more about that. In addition, we have heard, of course, of the agglutinins and other antibodies which are present in the blood serum.

Bloomfield at Hopkins made some very interesting studies on the effect of tonsil removal. He found in nurses examined at that institution that those who had their tonsils out had fewer throat infections than those who did not. He found, furthermore, that those nurses who carried streptococci in the tonsils were more immune from infection than those that did not. In other words, the presence of these bacteria in the tonsils in some way set up an immunity, so that it is not a bad thing to have a few bacteria about your person.

On the anatomic ground, the importance of the nasopharynx has been stressed, and this is a region which we do not always examine, because sometimes it is difficult. But you can avoid a great deal of embarrassment by making a routine examination of the nasopharynx in every case and not allowing a malignant growth or some disease about the mouth and the eustachian tube to escape your attention.

I could go on talking a long time about three papers, but I know that the hour is growing late, and I thank you for your attention.

DISCUSSION

Dr. E. M. Josephson, New York: In considering the conservative treatment of the various ills which we deal with in otolaryngology, we must bear in mind that there are many phases of the physiology of the mechanism that we are just recently becoming acquainted with. The excellent work being done in Dr. Dean's department down at George Washington University, for instance, is opening up new fields in the concept of the physiology of the mechanism of the upper respiratory tract. In the work we have been shown on the action of cilia, I don't think Dr. Proetz stressed the significance of calcium in ionized form, in the secretions of the upper respiratory tract for continued action of the cilia. It was Wenner, I think, who showed that ionized calcium is absolutely essential for physiological action of the cilia.

In our discussion of the tonsil the other day, there was one rather significant phase omitted, which hooks up with this problem of calcium in its relation to the function of the membranes of the upper respiratory tract.

In some work on colds associated with leukonychia, which I think I mentioned here last year, we have found, on continued study, that this type of cold, which is fundamentally a disturbance of the calcium metabolism, one frequently finds the accumulation of calculi in the tonsils of children. One expresses them on one day; the following day one can express still more. They accumulate very rapidly.

It seems to indicate that there is one function or dysfunction we may have overlooked, the elimination of calcium and other minerals in the presence of alkalosis or excess alkaline reserve. One of the things that is very helpful in treating that type of case, which generally shows continuous colds throughout the seasons, is the medicament which Wenner used experimentally, cortine.

In these cases, tonsillectomy is conservative therapy. The formation of concretions and calculi in the tonsil do not constitute the problem they once did. If one looks over the earlier literature of medicine, in the field of otolaryngology, one finds as early as 1549 references to repeated calculus formation in the tonsils, and in later literature one finds references quite numerous, which strikes one as quite a contrast with our recent lack of experience, such that many otolaryngologists have not seen a calculus in the tonsil.

Instructional Subjects

The American Academy
of Ophthalmology and Otolaryngology

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Thirty-Ninth Annual Meeting
Chicago . . . 1934

Instructional Subjects

DR. HARRY S. GRADLE, *Secretary for Ophthalmology*
DR. WILLIAM V. MULLIN, *Secretary for Otolaryngology*

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HISTOPATHOLOGY OF THE EYE

DR. HALLARD BEARD, Chicago
DR. J. S. FRIEDENWALD, Baltimore
DR. R. C. GAMBLE, Chicago
DR. THEODORE L. TERRY, Boston
DR. GEORGIANA DVORAK THEOBALD, Chicago

HISTOPATHOLOGY OF THE EAR, NOSE AND THROAT

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DR. LEROY A. SCHALL, Boston
DR. GREGOR MCGREGOR, Montreal

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DR. S. JUDD BEACH, Portland, Maine
Refinements of Refraction

DR. A. L. BROWN, Cincinnati, Ohio
Defects in Central Vision Field other than Glaucomatous

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Management and Surgery of Traumatic and Complicated Cataract

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Use of the Magnet

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Vernal Conjunctivitis. Clinical Aspect. Differential Diagnosis. Complications. Treatment

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DR. CHEVALIER L. JACKSON, Philadelphia, Pa.
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Management of Sinus Disease in Children

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Immediate Care of Head Injuries
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Benign Laryngeal Lesions

DR. FLETCHER WOODWARD, Charlottesville, Virginia
Management of Recent Fractures of the Nose

Presentation of Instruments

The American Academy
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Thirty-Ninth Annual Meeting
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Instruments and Appliances

Section on Ophthalmology

SUPRARENIN JELLY

BENNETT Y. ALVIS, M.D.

ST. LOUIS

CONCENTRATED solutions of Suprarenin have several objectionable features that are avoided by this preparation, which can be made up by any skillful pharmacist.

Glaukosan (links), as originally introduced by Hamburger for the treatment of glaucoma, is a foreign product so expensive and unstable as to be impractical for home use and limited in office practice by its cost and inconvenience.

Suprarenin bitartrate solutions, as described by John Green, are also unstable and quite expensive.

The method of preparation presented here was worked out by Mr. Roy Weidle of the Knight Drug Company in St. Louis, according to my suggestion, and has the following advantages:

1. *Stability* — A small tube prepared in February, 1934, is still effective and practically unchanged in appearance. It requires no especial care other than capping of the tube when not in use.
2. *Ease of Application* — It is easy to deposit a small amount of the jelly in the sac, either for the oculist, the patient, or a member of the family.
3. *Effectiveness* — The jelly remains in the sac for a prolonged period while the drug is slowly absorbed. The eye will remain blanched for a period of one to three or four hours.

4. *Lack of Reaction* — The nervousness and feeling of faintness so often experienced by the patient after the use of concentrated epinephrin solution is not so marked after use of the jelly, presumably due to slower absorption.

5. *Economy* — A one-dram (4.5 cc.) tube of a 2% jelly or a two-dram (9.0 cc.) tube of 1% costs the druggist about $0.75 and the patient $1.50 to $2.00, but with care this will supply 2 or 3 times a day application for several weeks and in office practice, even in a busy clinic, should last several months, since no large percentage of cases are suitable for its use.

Illustrative Cases:

Mr. W. T.; age 60; Chronic Glaucoma; trephining, cataract extraction, iris inclusion still has tension ranging up to 52 (Schiotz) when untreated. Not controlled by mioties. Four months ago began using 1% Suprarenin jelly 2 or 3 times daily. Tension range 22 to 27. Seen recently in the office after his supply was exhausted and after a trying journey in a subacute attack with tension of 52. Three hours after one instillation of the jelly, the globe was white, pain gone, tension 33 (Schiotz). Vision of 15/200 unchanged in four months.

Mr. G. D.; age 48; uveitis with secondary glaucoma; numerous posterior synechiæ. One per cent Suprarenin jelly dilated the pupil, freed the synechiæ, and kept the tension under control until the uveitis subsided.

R

	gm. or cc.
Suprarenin bitartrate	0.09
Gel of tragacanth	4.50

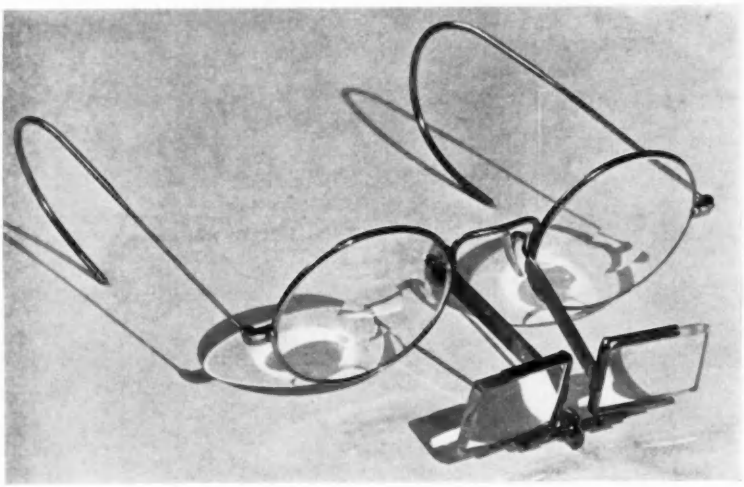
M. and place in flexo tube.
Sig.: Use in eye as directed.

Prepare the gel by soaking gum of tragacanth in water q.s. to make a medium thin jelly. Add phenol 0.5% as a preservative. Dissolve the suprarenin crystals in a few drops of water on the slab and mix thoroughly with the jelly.

A TELESCOPIC OPERATING LOUPE

BENNETT Y. ALVIS, M.D.

ST. LOUIS



THIS LOUPE is designed primarily for presbyopic eye surgeons who may feel the need of some magnification for the finer and more exacting bits of operative technic.

It provides magnification and at the same time gives a greater distance between the objective and operative field than is possible with the Beebe or Berger loupe of equal magnifying strength.

It consists essentially of the operator's corrective lenses before which is mounted the elements of a short Galileo's telescope. The plus objective lenses are mounted some convenient distance in advance of the spectacle frame while the minus element is applied to the front surface of the regular lenses either by cementing a small concave lens to its surface or by grinding a concave facet into its substance.

By increasing the distance between the lenses the refraction of the optical system is increased, shortening the working distance.

By increasing the strength of the minus element, while the distance between the lenses and the strength of the convex objective remain constant, the working distance is increased and magnification is decreased.

The instrument shown here is constructed on a pair of bifocal spectacles; the right lens being a minus four diopter cylinder at axis 75, the left a plus one and a half diopter cylinder at axis 105, reading addition of one and one-fourth diopters. The facet is minus three, objective plus eight, separation forty millimeters. This gives about 1.6 magnification with working distance of 188 mm. or 7½ inches.

The advantages of such a loupe are obvious.

1. The distance vision is clear above the central facet.
2. Bifocal segment permits selection of instruments or general manipulations at the regular working distance.
3. The binocular telescopic field is in central line of fixation at a convenient working distance.

Eyepiece	*Working Distance*	*Magnification*
	Objective = + 8.00 D.	
	Distance between Objective and Eyepiece = 40 mm.	
2.00 D.	162 mm.	1.647 ×
3.00 D.	188 mm.	1.596 ×
	Objective = + 12.00 D.	
	Distance between Objective and Eyepiece = 40 mm.	
8.00 D.	166 mm.	2.000 ×

I wish to acknowledge my indebtedness to Albert Aloe, Inc., of St. Louis, who made up this first loupe, and to Mr. J. C. Copeland of Riggs Optical Co., who furnished the table of calculated values shown here and who is making up several sample loupes and showing them at this meeting.

A NEW AND IMPROVED OPHTHALMOTROPE

WENDELL L. HUGHES, M.D.

HEMPSTEAD, N. Y.

A MODEL to demonstrate

1. The movements of the eyes.
2. The deviations of the eyes, vertical and horizontal, and the operations to correct them.
3. The demonstration of the retraction syndrome.

A motion picture film demonstrating the model is available and will be sent for the asking.

SOBAŃSKI'S NEW OPHTHALMODYNAMOMETER

HANS LAUBER, M.D.

WARSAW, POLAND

THE TWO existing types of ophthalmodynamometer have been devised by Baillart and by Baurmann. The first is easy to handle and strong; it is, however, not sensitive enough, especially for taking low degrees of pressure. The Baurmann apparatus is more sensitive, but it is tedious in manipulation and allows measurement of tensions up to 85 mm. mercury only. In working on intraocular venous pressure, the assistant at my clinic, Dr. Sobański, has constructed a new type apparatus, which is very subtle and, therefore, fit to measure accurately very low degrees of vascular pressure; it is simple in manipulation and enables us to measure pressures up to the height of 160 mm. mercury.

This apparatus, made completely of glass, consists of a cylinder with a piston gliding in it. The end of the piston is slightly concave, resembling the foot of the Schiötz tonometer. The cylinder continues into a tube with a three-way cock leading to a manometer. The cock allows connection of (1) the cylinder with the atmosphere, (2) the cylinder with the manometer, and (3) the manometer with the atmosphere. The manometer consists of a tube of 1 mm. diameter, with a millimetric division from 0 to 55.

In the tube there is a column of mercury, and on the side opposite the cylinder a metal rod 5 mm. long. Between the three-way cock and the manometer there is a swelling of the tube (reservoir I), separated from the manometer tube by a narrower neck of 0.5 mm. in diameter, so the mercury cannot easily glide into the reservoir. At the further end of the manometer are placed two enlargements (reservoirs II and III), between which a two-way cock is inserted. At the end of the manometer tube, opening into reservoir II, a platinum wire pre-

vents the metal rod from gliding into the collector. If the cock D is open, reservoirs II and III are in connection with the manometer. One can then measure pressures from 0 to 80 grams. The position of the mercury at 2 mm. equals a pressure of 5 g.; 5 mm., 8 g.; 10 mm., 16 g. Every to and fro movement of the mercury column by 5 mm. corresponds to 8 g. difference water.

In table 2, the corresponding pressures are noted in millimeters of mercury. By closing cock D, and thus interrupting the communication between reservoirs II and III, one produces a change in the value of the manometer division, so as to be able now to measure pressures up to 160 g.

The graph on table 3 shows how the movements of the mercury column in low pressures get very small. By slightly lifting the cock and moving the piston, the position of the mercury column can be regulated.

When the ophthalmodynamometer is not in use, the piston should be pushed completely into the cylinder, which must be shut off by cock C, the manometer being in connection with the atmosphere.

Before beginning measurements, the operator must (1) connect the cylinder with the atmosphere by turning cock C, (2) pull out the piston to 0.5 cm., (3) turn the cock so as to establish connection of the manometer with the atmosphere, (4) see that the mercury column is at 0.

The vascular pressure is taken by applying the piston on the globe over the tendon of the external rectus, holding the ophthalmodynamometer horizontally and perpendicularly to the surface of the globe, observing at the same time the fundus with an electric ophthalmoscope in the direct image. By pressure upon the globe, the mercury column moves towards reservoirs II and III.

When the necessary pressure has been attained, the pressure on the globe is slowly released; the mercury column returns to 0, and the metal rod remains on the spot it had occupied during the highest pressure reached, so one can read the divisions of the scale. By holding the ophthalmodynamometer vertically, the metal rod is made to join the mercury column. If pressure is exerted too long, the air can partly escape from the cylinder.

The pressure in millimeters of mercury is found in tables 2 and 3, which have been devised by using the tables of Baillart and Magitot.

The superiority of Sobański's ophthalmodynamometer consists in the possibility it gives of measuring both low and high pressures, a thing not possible with Baillart's or Baurmann's instruments. A further advantage is the moderate price of the apparatus; it is made under control of Dr. Sobański by the Physical Institute of the University of Warsaw.

APPARATUS FOR PROJECTING LIGHT STIMULI IN FIELD WORK

HANS LAUBER, M.D.

WARSAW, POLAND

A NUMBER OF contrivances have been constructed for casting luminous dots of perimeter or campimeter. Let me mention Zeemann (1925), Best (1930) whose apparatus has been made by Zeiss, Lopez Lacarrère (1931), Evans (1932), Magitot (1933). All these contrivances are either built for use in perimetric (Zeemann, Evans, Magitot), or campimetric work (Best, Lopez Lacarrère).

The apparatus I am showing here can serve for all purposes of field work. It consists of a small projecting apparatus with a lamp of low voltage as light source (5 volts, 6 amperes). The light supply is regulated by a rheostat. Provision is made for concentration of the bulb by screws.

First, in the tube containing the optic systems, there lies a double condenser, which can be moved fore and aft by means of a snailscrew. Then follows an iris diaphragm and a teleobjective. The latter projects an image of the diaphragm opening at any distance desired. The size of the diaphragm opening can be between 1 and 10 mm.; consequently, images between 1 and 10 mm. in diameter can be obtained on the screen. A number of colored glasses and a neutral-gray glass allow the operator to employ colored light for field work. The gray glass reduces the luminosity for work at short distance, f. i., in the Lloyd stereocampimeter. For work at short distance (stereocampimeter or perimeter), the negative lens of the teleobjective is taken out of the tube. If it is inserted again, the apparatus can serve for distances up to 2 meters.

Mr. Twaroch of Riechert Optic Works (Vienna) has made the necessary photometric measurements and calculations which

prove that the luminosity of the projected light spots at a distance of 2000 mm. is greater than the one of white paper illuminated by a 200-watt bulb. The smaller the distance of the projection, the more intense the light. For this reason, it is necessary to make use of the gray glass for small distances of projection, in order to prevent irradiation.

The apparatus is light enough to be wielded with one hand, and, consequently, can be employed for stereocampimeter, perimeter and Bjerrum screen. It possesses this advantage in addition to those of the other contrivances mentioned above.

CHROMATIC MALINGERING TEST CHARTS AND MODIFICATIONS OF THREE OTHER MALINGERING TESTS

R. E. Mason, M.D.

ST. LOUIS

In times of industrial depressions, the oculist is confronted with an increasing number of patients who make exaggerated claims as to the mount of vision lost following an alleged injury, frequently claiming a complete loss of some function of the eye, which, after careful examination, we find to be normal. These examinations are medico-legal in nature and must necessarily be as accurate as possible and the report must state the efficiency of the eye in percentages. To facilitate the examination, the writer has designed a malingering test which he wishes to present, and modifications of three other malingering tests, which, in his opinion, enhance their value.

The chromatic malingering charts are constructed and used as follows:

All Snellen-type sizes, from the 20/250 to the 20/20, are accurately reproduced by a commercial artist on separate boards for each size of letter, handpainted in alternate red and green

shades, the artist using the red and blue-green lenses which are found in the regulation test cabinet for his determinations. The aim is to get such shades of red and green as will completely fade out when viewed through the red and blue-green lenses. In the construction of the green letter, it was found that it must be painted on a light grey background in order to fade out completely when seen through the blue-green lens. The test is used on the regulation 20-foot range and the letters are placed separately in an artificially-lighted test cabinet.

The advantages of this test are that it is new to the expert malingerer and is conclusive in its result. Another great advantage is that it registers the visual acuity of the patient examined. Concerning the visual acuity, however, the writer has found that the colored letters, being in pastel shades, vary somewhat in visibility from the regular Snellen black letters on a white card, in that a person with normal vision who reads 20/20 on the regulation Snellen chart, will see only a shade better than 20/30 on the chromatic malingering chart. This difference must be taken into account in estimating the visual acuity.

The test is conducted in the following manner. The patient to be examined is placed on the test range and a trial frame adjusted. The nurse has been instructed to place in the test cabinet, for example, the 20/30 chromatic card, not allowing the patient at any time to see the card before the test. With an alleged injury in the right eye, a plano lens is placed before this eye and the red lens before the left eye, both eyes being closed at the time. We then ask the patient to open both eyes and place our fingers above each upper lid to make sure the eyes are kept open. The patient is then told to read the colored letters with both eyes open. Obviously, if he reads any red letters on the 20/30 group, he has admitted seeing them with his alleged injured eye and his vision should be registered as 20/30 + in the injured eye. The same test is repeated with the green letters. It has never been found feasible to use the red and green lenses at the same time.

Under the Workmen's Compensation Laws of most states, the near vision of each claimant at a distance of 14 inches must be reported. Greater weight is attached to a loss of near vision than of distance vision. With this in view, I wish to present a malingering test for near vision only, which is not

entirely original with me, but is a modification of a test, which I understand was first introduced by Dr. Judson Harman. The apparatus consists of a board 14 inches long, to which is attached a handle. At the distal end is an upright board to support a near vision reading card. Five inches from the distal end there is another board on hinges, so that it may be upright or lie flat, which has a round opening in the center near the top about 1¼ inches in diameter. When the board is placed against the patient's chin and held firmly, the reading card being in position, the patient with both eyes open is asked to read, looking through the round opening of the middle board. It is necessary that the examiner be certain that the patient keeps both eyes open continually. On account of the interpupillary distance and the visual angle, it is obvious that the patient will read letters or words farther to the left with his right eye and farther to the right with his left eye. His visual acuity for near is thus easily determined. I designed this board at 14 inches because this meets the requirement of the near vision test for compensation courts. Finding that bar-reading was also a very useful test for visual acuity for near, I had this instrument constructed so that the middle board could be let down flat on its hinges and a wooden peg 5 inches long inserted 1 inch proximal to this board. This makes a stationary and convenient bar-reading test. In difficult cases, I also resort to near

vision cards which are printed with words in alternate black and red type, using a red lens over the uninjured eye. The test is then carried out in a manner similar to that used in the distance test with both eyes open. Words that are pronounced that are printed in red must necessarily have been seen with the alleged injured eye.

Finding that it is often necessary to have recourse to more than one malingering test for distance, I have modified, and I think improved, the old prism test. Briefly, it is as follows: With a patient on the 20-foot range, and a separate line of Snellen-test letters in the test cabinet, we ask the patient to close his alleged injured eye; we then hold a 5 degree prism, base up, in front of the uninjured eye with the base of the prism intersecting the center of the pupil. This will immediately give him monocular diplopia and he will admit seeing two lines of the Snellen-letters with his uninjured eye. When this idea is fixed in his mind, we ask him to open both eyes, at the same time we advance the base of the prism upward across the pupil

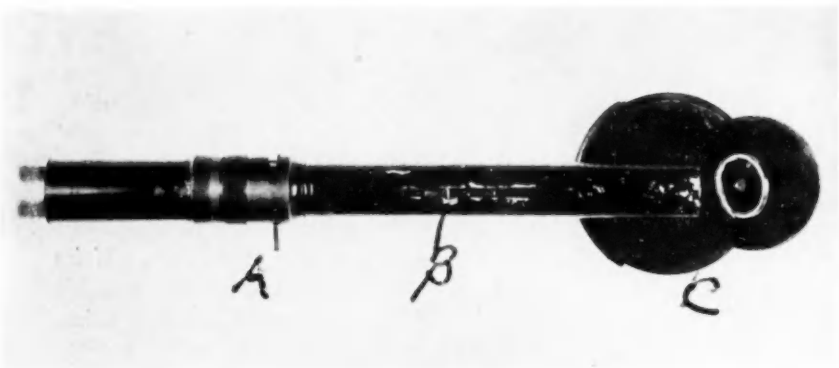
of his uninjured eye and again ask him to read the two lines of Snellen-letters. If he complies with this request and states that he sees both lines, he has admitted binocular diplopia with equal vision in both eyes. The improvement we made on this test was having the prism ground in the form of a rectangle, having square edges and mounted with a handle for more convenient use.

Another near vision test which we modified, but is not original with us, which we use as a near vision test on a patient not of presbyopic age and nearly emmetropic in his uninjured eye is that of a reversible spectacle frame constructed so as to contain on one side a + 3.00 diopter sphere and on the other a plano lens. With the frame adjusted on the patient with the 3.00 diopter sphere in front of the uninjured eye, the patient is requested to read the reading card held at a distance of 6 inches in front of his eyes. Both eyes are open. With one normal eye the patient will easily comply with this request. After a short interval, in which an adjustment of the frame is made, the same card is again held in front of the patient at a distance of 14 inches and he is requested to read the same lines. If he again reads the lines correctly, it is evident that he has used his alleged injured eye as the card is now beyond his near point.

COMBINATION OPHTHALMOSCOPE, ASTIGMANOMETER AND AXIS FINDER

HARVEY B. SEARCY, M.D.

TUSCALOOSA, ALA.



THE SLIDE A has one large hole through which the light is used when the instrument is used as an ophthalmoscope. Also a small hole of pinpoint size for determining the astigmatism.

B is a battery of lenses which makes the rays of light from the little hole in A parallel when the lenses are in the central position.

C is an ordinary ophthalmoscope head.

The instrument is used in the same position as an ophthalmoscope with the large hole of A in position and the fundus studied. The battery of lenses, B, may be pushed up or down in its tube making the ray of light parallel, convergent or divergent, giving a bright spot upon the retina or a more diffuse illumination of the retina, as desired.

The small pinpoint hole is pushed over in place, the instrument still being held in position for ophthalmoscopy and the pinpoint of light is focussed upon the retina; when properly focussed

it is a perfect small pinpoint of light and as the battery of lenses, B, is pushed up or down this pinpoint of light becomes more diffuse. If there is any astigmatism the pinpoint of light will become elongated and then finally a long streak across the retina indicating accurately the axis of astigmatism. Cylinders may be placed in a trial frame in front of the patient's eye and this elongation or streak neutralized, giving the exact astigmatic error and axis, when it again becomes a round diffuse light or pinpoint. The refractive error may be estimated by the position of the battery of lenses, B, in the tube, to which is to be added the astigmatism lenses found.

HAND MICROSCOPE AND SLITLAMP

HARVEY B. SEARCY, M.D.

TUSCALOOSA, ALA.

A DE ZENG ophthalmoscope handle is used, the head having been removed, and a 10× magnifying loupe added. The sliding battery of lenses will focus the beam of light, i.e., the filament of the light bulb, giving the slit effect of a slitlamp of 10× magnification. Also the sliding battery of lenses will give a diffuse light suitable for studying the cornea, iris, etc. It is light and easily held in the hand. Also a 20× loupe is used similarly.

JAEGER TEST TYPE

HARVEY B. SEARCY, M.D.

TUSCALOOSA, ALA.

THE WORDS "one," "two," etc., are used indicating the size of type. These few words eliminate the time taken in refraction usually consumed by the patient in satisfying his curiosity as to what is written upon the chart. Upon the other side of the card is the word "six" written the size of Jaeger 10. This one word gets the patient to concentrate much better and facilitates refraction.

OCULAR ENDOSCOPE

(AN INSTRUMENT FOR THE REMOVAL OF INTRAVITREOUS NON-MAGNETIC FOREIGN BODIES)

HARVEY E. THORPE, M.D., F.A.C.S.

PITTSBURGH

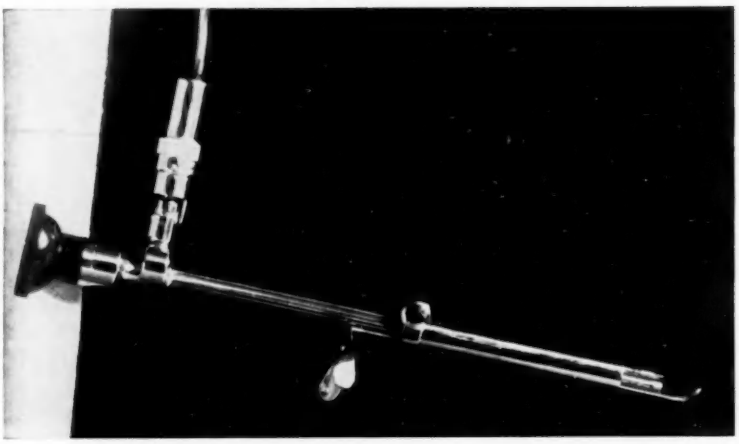


FIG. 1 — (Thorpe) — Self-luminous Ocular Endoscope.

THE REMOVAL of intraocular foreign bodies has long attracted the attention of physicians. The utilization of the electromagnet has aided in the partial solution of this problem.

The extraction of non-magnetic intravitreous foreign bodies such as brass, lead shot, wood and larvæ has, however, been beset with particular difficulties. The chief of these has been that the surgeon could not readily see the object he was trying to remove. With this view in mind, it seemed practical to devise an instrument combining a small telescope, a fine forceps, and a source of illumination.

A special inverted Galileon telescope, similar to those used in cystoscopes, pharyngoscopes and other endoscopes is utilized. This permits a view of a field 12 mm. in diameter at one inch distance. The telescope is 2½ mm. in diameter.

The shape and size of the intraocular object determines the type of jaws for the attached forceps, e.g., ring-shaped jaws for

BB lead shot, serrated blades for irregular-shaped foreign bodies. The portion of the forceps which passes through the sclera consists of a tube 1 to 1½ mm. in diameter. A scissors attachment may be used to cut vitreous bands by direct vision. The forceps is attached to the telescope and light carrier by means of an adjustable sheath. This also prevents the forceps from striking the opposite wall of the globe.

Illumination is obtained in three ways: (1) The non-luminous instrument is used with illumination through the dilated pupil by a May ophthalmoscope with the Recoss disc removed or by a hammer lamp. (2) Transillumination through the sclera may also be used with the non-luminous form of instrument. (3) The self-luminous form is the best and has a miniature lamp adjacent to the telescope, and is operated by a battery handle or by a cord from a rheostat.

The three integral parts of the instrument lie in one row, viz., lamp, telescope, and forceps (Fig. 1). The total distance across the instrument is 6 mm. A scleral incision 8 mm. long is required to admit the end of the instrument to the globe.

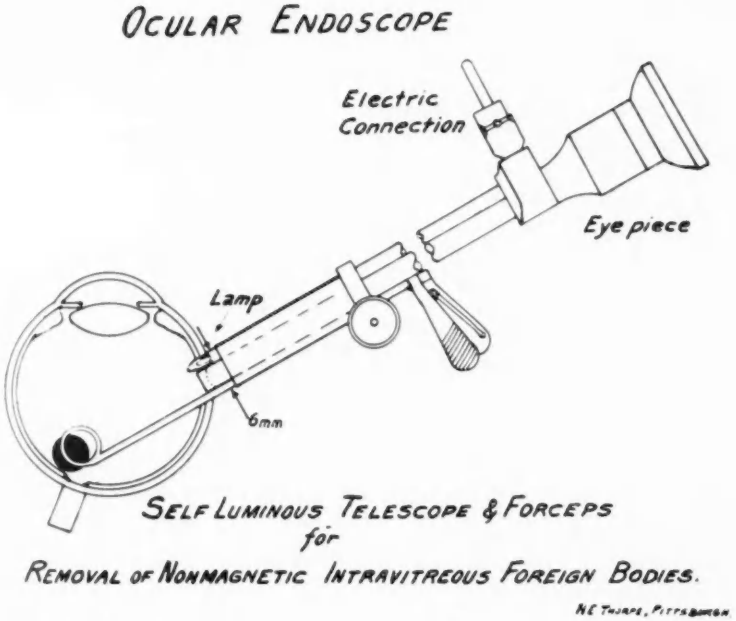


FIG. 2 — (Thorpe) — Method of Operation with Self-luminous Ocular Endoscope.

Xray and ophthalmoscopic examination are, of course, made before operation. The conjunctiva is dissected up and scleral sutures are inserted previous to its incision, which should be made preferably with a high frequency knife to avoid excessive bleeding. The incision in the sclera is anterio-posterior, preferably in the lower temporal quadrant, 8 mm. from the limbus. The self-luminous instrument is introduced in the eyeball through the scleral incision, the telescope stopping at the inner surface of the globe so as not to displace the vitreous humor (See Fig. 2). (The non-luminous instrument requires a 6 mm. scleral incision).

A foroblique telescope with a small lamp on its end is also being tried. This would permit a 6 mm. incision, but the insertion of the light into the globe will cause displacement of some vitreous.

With the aid of the Ocular Endoscope it is possible to have a direct view of the interior of the globe with a minimum manipulation of small instruments. This method minimizes the amount of guessing in the search for non-magnetic intravitreous bodies and facilitates their removal.*

* The model shown was manufactured by American Cystoscope Makers, Inc., New York, N. Y.

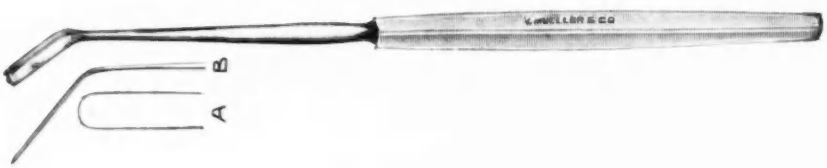
Instruments and Appliances

Section on Otolaryngology

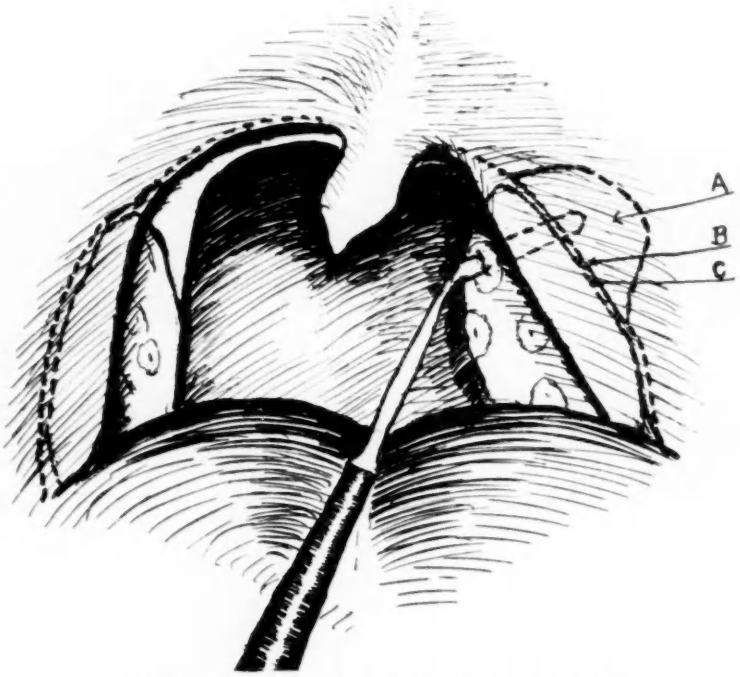
NEW KNIFE FOR OPENING PERITONSILLAR ABSCESS

R. G. Reaves, M.D.

KNOXVILLE, TENN.



For the past fifteen years I have opened all peritonsillar abscesses through the upper pole of the tonsil by the following method: I first take a probe and gently locate a crypt which reaches to the capsule of the tonsil. After having located the crypt and decided whether I should point the knife directly outward or slightly upward, backward or forward, I then pass the knife back to the capsule, telling the patient it is going to hurt, pop the knife into the abscess, then an upward and downward motion and out. I now take a curved hemostat and gently place it into the cavity, open same and let the pus out. Having used this method so long, and having always found it satisfactory, I have decided to offer a specially-designed knife for this operation. The knife, as shown in the illustration, is of such an angle to make it easy to enter the crypt. It is double-edged, cutting both up and down. The end is sharp, but not pointed, making it easy to pass back to the bottom of the crypt without entering into the tonsil tissue.



A, Abscess; *B*, Fascia; *C*, Tonsillar Capsule (Reaves).

A study of the anatomical location of a peritonsillar abscess should convince one that this is an easy approach to the abscess. *First*, on removing a tonsil you will always find a crypt near the upper pole that will permit a hemostat to pass to the thin capsule of the tonsil. *Second*, anatomical location as described in *Operative Surgery:* "This is an abscess usually situated in the supratonsillar fossa in a space bounded below by the tonsil, externally by the superior constrictor, in front by the palatoglossus and behind by the palatopharyngeus muscle, and above by the angular space formed by the union of these muscles with the superior constrictor. The abscess is external to the tonsil." The superior constrictor muscle is pushed outward, the tonsil together with the fascia of the tonsillar fossa are pushed inward. The knife has to pass through only the tonsil capsule and fascia to enter the abscess. It gives a sensation of popping through a membrane into a cavity.

Xray picture with instruments in the crypts of the tonsils showing that the crypt reached to the capsule of the tonsil (Reaves).

Advantages: (a) Hits the abscess almost 100%. (b) Can be done even if patient tries to jump away. (c) Eliminates probing for pus. (d) Very little bleeding, as a rule.

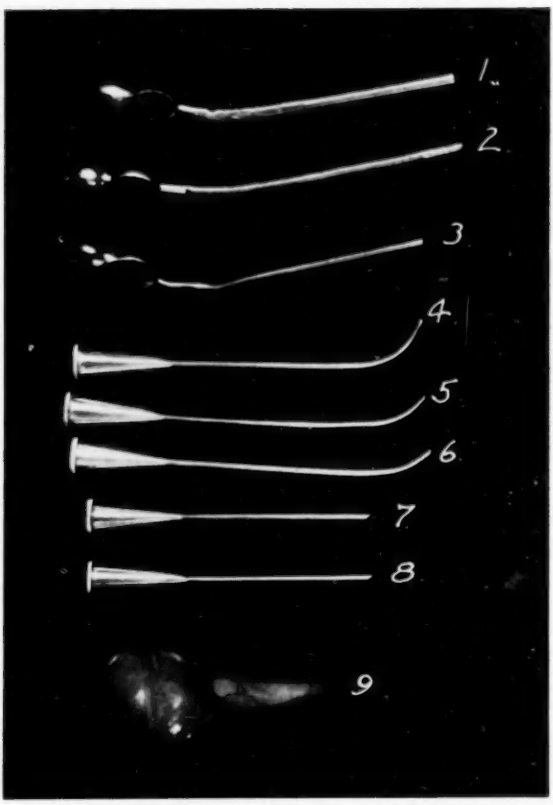
I have never had much bleeding in this method. As a matter of fact I feel it is very safe because the abscess has pushed the elected point away from the larger vessels.

If you are satisfied with your method, fine! If not, try this.

SUCTION TIPS, STRAIGHT TROCARS AND ANTRUM WASHING CANULA

HARVEY B. SEARCY, M.D.

TUSCALOOSA, ALA.



Nos. 1, 2, and 3 are Suction Tips; Nos. 4, 5, and 6 are Antrum Washing Canula; Nos. 7 and 8, Straight Trocars; No. 9, Rubber Syringe.

The suction tips are used in nose work to remove blood, pus, polypi and tissue after being cut off.

In using suction for sinus infection a cotton plug saturated with argyrol or some antiseptic astringent with high osmotic tension is placed as near the sinus orifice as possible and left in place for 10 minutes, more or less. Suction is then applied with a suction tip pressed against the cotton plug and capillary attraction will fill the cotton plug with the pus to be removed. Repeat until no pus is obtained.

In using suction to clean discharging ears, fill the ear with a cigarette drain of cotton well saturated with some antiseptic solution and apply the suction tip to the external end of the cotton drain and capillary attraction will fill the cotton with the discharge and when removed the ear will be cleaner, repeat until no pus is removed; in this way there is no danger of too much suction in the middle ear, etc.

Suction in blepharitis is done by softening the scales upon the lashes with vaseline (borated), then massaging the lashes, using suction at the same time, removing the scales and evacuating the follicles. This is more efficacious, and accomplished with less discomfort, than when massage is done with the fingers, glass rod, etc.

The very large flange permits the use of a soft rubber syringe with the antrum washing canula and straight trocars, even when the syringe is soft and old.

In washing the antrum and other sinuses, I always use oil, usually chlorotone inhalant ⅓, and liquid petrolatum ⅔. As has been shown in recent literature, oily solutions inhibit the ciliary activity of the epithelial cells and therefore the oily solutions remain in place longer. The menthol, etc., causes the natural orifices to remain patent, the odor is masked or relieved, and the pain following the antrum washing is greatly lessened.

The soft rubber syringe is partially filled with the oil, air is injected slowly at first, the squeezing of the syringe is continued and the oil follows the air; the syringe is removed from the canula or trocar and allowed to fill with air and the head is so held as to empty the antrum when air is again introduced. Some of the soothing antiseptic oil is not removed, but clings to the membranes of the sinus.

Minutes

The American Academy
of Ophthalmology and Otolaryngology

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Thirty-Ninth Annual Meeting
Chicago . . . 1934

MINUTES

AMERICAN ACADEMY *of* OPHTHALMOLOGY AND OTOLARYNGOLOGY

MONDAY, SEPTEMBER 10, 1934, 9:45 A.M.

Joint Session

THE OPENING SESSION of the Thirty-Ninth Annual Convention, American Academy of Ophthalmology and Otolaryngology, held at the Hotel Sherman, Chicago, Illinois, September 10-14, 1934, convened at nine-forty-five o'clock, Dr. John M. Wheeler, New York, President, presiding.

PRESIDENT WHEELER: The Thirty-ninth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology is now officially opened.

I first introduce Professor Hans Lauber of Warsaw, Poland, whom many of you have known as a teacher, but whom I especially introduce as a friend, Professor Lauber.

PROFESSOR HANS LAUBER: Mr. President, Ladies and Gentlemen: I highly appreciate this extraordinary honor of being invited here to speak before such a distinguished society as the American Academy of Ophthalmology and Otolaryngology. I have met old and made new friends in this country, and my heart feels very warm from the contacts.

Let me join in the wish of our President, that this meeting shall be a very successful one and that you will remember it in future years as a very enjoyable session.

PRESIDENT WHEELER: Dr. Novak, will you make the announcements?

Dr. Frank J. Novak, Jr., Chicago, Third Vice President and General Chairman of the Chicago Convention, called the attention of the members to the Teachers' Section Dinner, Academy's Dinner Dance, and the Golf Party.

PRESIDENT WHEELER: Former presidents of the Academy have discussed, in masterly fashion, many important subjects, but so far no one has offered material relative to the blind individual.

President Wheeler then read his address, "Blind People."

PRESIDENT WHEELER: We will now have the address of our guest of honor, Professor Lauber, who will speak on "Diagnostic and Prognostic Importance of Ophthalmoscopy in Red-Free Light, with Special Reference to the Affections of the Optic Nerve and the Chiasma."

Professor Hans Lauber read his address.

PRESIDENT WHEELER: The program continues with a Symposium on Exophthalmos. The first speaker in this symposium is Dr. Russell Wilder, Rochester, Minnesota.

Dr. Wilder read the paper, "Constitutional Factors," prepared by Dr. William A. Plummer, Rochester, Minnesota, and himself.

PRESIDENT WHEELER: The order of the next two papers will be changed, and Dr. Reese will now speak on "Exophthalmos Caused by Lesions of the Eye and Orbit."

Dr. Algernon B. Reese, New York, read his paper.

PRESIDENT WHEELER: The last paper on the program this morning is by Dr. Eagleton, who will speak on "Exophthalmos Caused by Lesions of the Nasal and Cranial Cavities."

Dr. Wells P. Eagleton read his paper.

MONDAY, SEPTEMBER 10, 1934, 2:15 P.M.

Section on Otolaryngology

The meeting convened at two-fifteen o'clock, First Vice President LeRoy A. Schall, Boston, Mass., presiding.

CHAIRMAN SCHALL: Will the meeting please come to order? The first item on the program this afternoon is "Report on Labyrinth Work" by Dr. W. J. McNally, Montreal, Canada.

Dr. McNally gave his report.

CHAIRMAN SCHALL: Dr. McNally's presentation is a report upon his work in the labyrinth, therefore, it is not open for discussion.

Dr. Charles H. Watkins, Rochester, Minn., read a paper entitled, "Angranulocytic Angina Following Ingestion of Amidopyrine or the Barbiturates." Discussed by Drs. John J. Shea, Memphis, Tenn., and Gordon B. New, Rochester, Minn.

Dr. Albert D. Kaiser, Rochester, N. Y.: Mr. Chairman, Ladies and Gentlemen: I greatly appreciate the honor of being able to participate in this symposium on the tonsil problem. We in the field of medicine, pediatrics and general medicine, have very much in common. We have the same patients in our hospital and private practice, and it seems quite fitting that at a gathering of this sort these problems should be discussed.

My own experience has been entirely with children and any statements that I make apply only to children up to the age of adolescence, or perhaps sixteen years of age.

Dr. Kaiser then read his paper, "Why and When the Tonsils Should Come Out."

Chairman Schall: Among the hospitals of Boston, there is one that is somewhat unique, unique in the fact that by the will of its founders it was established to take care of patients with incurable or chronic disease. About 80% of the patients that have gone to this institution are arthritics. The average duration of these patients in the hospital is one year. Many of the patients have been there for fourteen to sixteen years.

About three years ago one of the internists of this hospital decided to find out what has been the life course, what has been the benefit to these patients with chronic disease, and he set about to trace down their life story. He has burned the midnight oil. He has traveled miles chasing these patients through other institutions, through the autopsy rooms. He has found that 100 of them have died; 30 of them have been autopsied. For three years he has collected and reconstructed the medical life story of some 700 patients with chronic disease.

We have turned to this man to tell us about his findings and his opinion as to the tonsils as a factor of systemic infection, Dr. Nissen.

Dr. H. Archibald Nissen, Boston, Mass., read his paper, "Tonsils as Foci of Systemic Infection."

Chairman Schall: We will now take the symposium away from our colleagues and turn it back to the laryngologists. The title of the next paper is "Preparation and Management of

Patients Previous to and During Tonsil Operation," by Dr. David H. Jones of New York.

DR. JONES: Mr. Chairman, Ladies and Gentlemen: I thank you very much for the honor you have conferred upon me in allowing me to present this paper to you.

Dr. Jones then read his paper.

CHAIRMAN SCHALL: The summation of these papers will be given by Dr. Mosher.

Dr. Harris P. Mosher, Boston, Mass., read his summation.

CHAIRMAN SCHALL: It was the plan of the Program Committee that this, being a symposium, the usual discussion would be omitted, and due to the lateness of the hour, I think it best to follow that original plan. Therefore, I declare this session adjourned.

The meeting recessed at five o'clock.

MONDAY, SEPTEMBER 10, 1934, 6:00 P.M.

Teachers' Section

The Dinner Meeting of the Teachers' Section convened at six o'clock on Monday, September 10, 1934. Dr. Samuel J. Kopetzky, New York, Chairman, presided.

Opening remarks by Chairman Kopetzky.

Remarks by the President, Dr. Wheeler.

Remarks by the guest of honor, Professor Hans Lauber.

Report on Plan, Scope, and Organization of Newly Constituted Advisory Medical Council given by Dr. W. P. Wherry, Omaha, Nebr., Executive Secretary-Treasurer.

Address by Dr. George H. Meeker, Dean, Graduate School of Medicine, University of Pennsylvania, on "Trends in Graduate Education, with Reference to the Integration of Extramural Teaching with the University Curriculum."

Address by Dr. Raymond B. Allen, Associate Director, New York Postgraduate Medical School, Columbia University, on "University Standards in Training Ophthalmologists and Otolaryngologists."

Introduction of, and remarks by, the new Chairman of the Teachers' Section, Dr. A. D. Ruedemann, Cleveland.

OFFICERS ELECTED FOR 1935

DR. A. D. RUEDEMANN ..... *Chairman*
DR. GEORGE M. COATES... *Associate Chairman*
DR. RALPH A. FENTON ..... *Secretary*

Directors, 1 Year (term expires 1935): Dr. G. M. Coates, Philadelphia; Dr. A. D. Ruedemann, Cleveland. 2 Years (term expires 1936): Dr. L. W. Dean, St. Louis; Dr. J. H. Dunnington, New York. 3 Years (term expires 1937): Dr. A. C. Furstenberg, Ann Arbor; Dr. T. D. Allen, Chicago. 4 Years (term expires 1938): Dr. D. M. Lierle, Iowa City; Dr. F. H. Adler, Philadelphia. 5 Years (term expires 1939): Dr. S. J. Kopetzky, New York; Dr. F. E. Burch, St. Paul.

The meeting adjourned at ten-twenty o'clock.

TUESDAY, SEPTEMBER 11, 1934, 2:30 P.M.

Section on Ophthalmology

The meeting convened at two-thirty o'clock, President Wheeler presiding.

PRESIDENT WHEELER: The meeting will come to order. The first paper on the afternoon program is "Report on the Operative Results of 150 Cases of Detachment of the Retina," by Dr. John H. Dunnington and Dr. John P. Macnie, New York, Dr. Dunnington.

Dr. Dunnington read the paper prepared by himself and Dr. Macnie.

PRESIDENT WHEELER: May I call your attention to the time limits on papers and discussions? If I have it correctly, the time limit on the paper presented is fifteen minutes. The scheduled opener of the discussion has ten minutes, and other discussors three minutes. In this case, we have two chief discussors, and I should say we ought to divide ten by two, giving each one five minutes. Dr. Walker will now discuss this paper.

Dr. Clifford Walker, Los Angeles, Calif., read his discussion.

PRESIDENT WHEELER: The discussion will be continued by Dr. F. E. Burch of St. Paul, Minn.

Dr. Burch read his discussion.

PRESIDENT WHEELER: If my memory serves me correctly, about twenty years ago, our fellow member, Dr. Emmet L. Jones of Cumberland, Md., operated successfully on a case of detached retina by the cautery puncture method. I wonder if Dr. Jones will say a word and corroborate my statement?

Dr. Jones gave his discussion. Further discussion by Dr. Raymond Emory Meek, New York.

PRESIDENT WHEELER: Dr. Macnie, will you close the discussion on this subject?

Dr. Macnie closed the discussion.

PRESIDENT WHEELER: The next paper on our program is entitled "The Use of Sclerosing Solutions in Ophthalmic Therapeutics," by Dr. M. F. Weymann of Los Angeles.

Dr. Weymann read his paper.

PRESIDENT WHEELER: The discussion will be opened by Dr. Sanford R. Gifford of Chicago.

Dr. Gifford read his discussion. Discussion was continued by Dr. R. O. Rychener, Memphis, Tenn. Dr. Weymann closed.

PRESIDENT WHEELER: Our next paper is on "Suppression in Antipathy Toward Single Vision," by Dr. Hendrie W. Grant of St. Paul, and Dr. F. N. Knapp of Duluth.

Dr. Grant read the paper prepared by himself and Dr. Knapp.

Second Vice President W. F. Hardy, St. Louis, Mo., assumed the chair.

CHAIRMAN HARDY: Discussion of Dr. Grant's paper will be opened by Dr. L. C. Peter of Philadelphia.

Dr. Peter read his discussion.

CHAIRMAN HARDY: The discussion will be continued by Dr. John H. Dunnington of New York.

Dr. Dunnington read his discussion.

CHAIRMAN HARDY: I will ask either Dr. Grant or Dr. Knapp to close.

Dr. Knapp closed the discussion.

CHAIRMAN HARDY: The next paper on the program is "Divergence Paralysis Due to Head Injury," by Dr. Harry M. Weed of Buffalo, N. Y.

Dr. Weed read his paper.

CHAIRMAN HARDY: Dr. E. B. Dunphy of Boston will open the discussion on Dr. Weed's paper.

Dr. Dunphy read his discussion. Dr. F. H. Adler, Philadelphia, also discussed the paper. Discussion was closed by Dr. Weed.

CHAIRMAN HARDY: The final paper on the program today is "Detachment of the Internal Limiting Membrane of the Retina," by Dr. D. J. Lyle of Cincinnati.

Dr. Lyle read his paper.

CHAIRMAN HARDY: Dr. Wilfred Fry of Philadelphia will open the discussion on Dr. Lyle's paper.

Dr. Fry read his discussion.

CHAIRMAN HARDY: Discussion will be continued by Dr. F. B. Fralick of Ann Arbor, Mich.

Dr. Fralick read his discussion.

CHAIRMAN HARDY: Dr. Lyle, will you close, please?

Dr. Lyle closed the discussion.

The meeting recessed at five-forty o'clock.

TUESDAY EVENING, SEPTEMBER 11, 1934

Banquet

Dr. Carl Koller, New York, was honored by the Academy in commemoration of the fiftieth anniversary of his discovery of cocaine as a local anesthetic. The following telegram was read at the banquet:

Dr. John M. Wheeler, President
American Academy of Ophthalmology and Otolaryngology

Mr. Chairman and Members of the Academy: I am very proud to be thus honored by you and am happy to have lived to see the progress in the different branches of medicine and surgery

since I first made my modest experiments, exactly fifty years ago. It might here be the place to point out that it was not suggested to me by anybody, as has been stated; also, lately, that I should try the anesthetic properties of cocaine on the eye.

The anesthetic properties of cocaine had been known for twenty-five years to a host of physiologists and pharmacologists and were waiting for a man who was aware of the need of local anesthesia in the practice of medicine. The correct facts are related in a paper read before the International Congress of Anesthetists in Washington in 1927 and reprinted in the Journal of the American Medical Association in 1928.

I am grateful for the distinction which you thus confer on me and thank you from the bottom of my heart.

CARL KOLLER

THURSDAY, SEPTEMBER 13, 1934, 2:00 P.M.

Section on Otolaryngology

The meeting convened at two o'clock, First Vice President Schall presiding.

CHAIRMAN SCHALL: The meeting will please come to order. The Program Committee requests that you submit suggestions for next year's program. Every suggestion received will be given due consideration. Items relative to otolaryngological subjects should be addressed to Dr. Myers; those pertaining to ophthalmology, to Dr. Benedict.

The first paper on the program this afternoon is "Report on Sinus Mucous Membrane," by Dr. Olof Larsell and Dr. Ralph A. Fenton of Portland, Oregon. I am sorry to announce to the Academy Dr. Larsell's misfortune in losing an eye. The report will be submitted by Dr. Fenton.

Dr. Fenton read the paper prepared by himself and Dr. Larsell.

CHAIRMAN SCHALL: The second paper this afternoon is "Research Studies in Visualization of the Vascular Supply of the Head and Neck," by Dr. O. Jason Dixon of Kansas City, Mo.

Dr. Dixon read his paper.

CHAIRMAN SCHALL: The discussion of Dr. Dixon's paper will be opened by Dr. Samuel J. Kopetzky, New York.

Dr. Kopetzky read his discussion.

CHAIRMAN SCHALL: The discussion of Dr. Dixon's paper will be continued by Dr. Frank R. Spencer, Boulder, Colorado.

Dr. Spencer read his discussion.

CHAIRMAN SCHALL: There being no further discussion, I will call upon Dr. Dixon to close the discussion.

Dr. Dixon closed.

CHAIRMAN SCHALL: We have now reached our symposium on "The Conservative Treatment of the Nose, Throat and Ear." The first paper is on "Conservative Treatment of the Nose and Accessory Sinuses," by Dr. W. E. Grove of Milwaukee, Wis.

Dr. Grove read his paper.

Third Vice President Frank J. Novak, Jr., Chicago, assumed the chair.

CHAIRMAN NOVAK: The next paper in this symposium is "Conservative Treatment of the Pharynx," by Dr. John W. Carmack of Indianapolis, Indiana.

Dr. Carmack read his paper.

CHAIRMAN NOVAK: The next paper is "Conservative Treatment in Diseases of the Ear," by Dr. James A. Babbitt of Philadelphia.

Dr. Babbitt read his paper.

First Vice President Schall resumed the chair.

CHAIRMAN SCHALL: The discussion of these papers in this symposium will be opened by Dr. Samuel Iglauer of Cincinnati.

Dr. Iglauer read his discussion. Discussion was continued by Dr. E. M. Josephson, New York.

CHAIRMAN SCHALL: Is there any further discussion? If not, I will ask Dr. Grove to close.

DR. GROVE: I have nothing further.

CHAIRMAN SCHALL: Dr. Carmack? (Absent). Dr. Babbitt?

DR. BABBITT: Nothing further.

The meeting recessed at four-ten o'clock.

FRIDAY, SEPTEMBER 14, 1934, 9:10 A.M.

Section on Ophthalmology

The meeting convened at nine-ten o'clock, President Wheeler presiding.

PRESIDENT WHEELER: The meeting will come to order. The first paper on this morning's program is "Ophthalmomyiasis: A Review of the Literature and Report of a Case of Ophthalmomyiasis Interna Posterior," by Dr. William B. Anderson of Durham, North Carolina.

Dr. Anderson read his paper.

PRESIDENT WHEELER: Discussion will be opened by Dr. M. P. de Boe of Miami, Florida.

Dr. de Boe read his discussion.

PRESIDENT WHEELER: The discussion will be continued by Dr. Martha Lyon, South Bend, Indiana.

PRESIDENT WHEELER: Is there further discussion of this interesting subject? If not, Dr. Anderson, will you close the discussion?

DR. ANDERSON: I have nothing to add.

PRESIDENT WHEELER: The next paper is by Dr. A. C. Jones, Boise, Idaho, on "Oil Cyst of the Orbit with Carcinomatosis."

Dr. Jones read his paper.

PRESIDENT WHEELER: Dr. Gamble will open the discussion.

Dr. Richard C. Gamble, Chicago, read his discussion.

PRESIDENT WHEELER: The discussion will be continued by Dr. Mary K. Asbury, Cincinnati.

Dr. Asbury gave her discussion.

PRESIDENT WHEELER: Is there further discussion of Dr. Jones' paper? If not, Dr. Jones, will you close the discussion?

DR. JONES: I have nothing further to add, except that I want to thank Dr. Gamble and Dr. Asbury for their kind discussion of this paper.

PRESIDENT WHEELER: The next paper is on "The Relation of Right and Left Side Dominance to Strabismus," by Drs. Walter H. Fink and Bryng. Bryngelson of Minneapolis.

Dr. Fink read the paper prepared by himself and Dr. Bryngelson.

PRESIDENT WHEELER: Dr. Bryngelson.

Dr. Bryngelson read his discussion.

Dr. D. T. Vail, Jr., Cincinnati, read his continuation of the discussion. The paper was also discussed by Dr. Oscar Wilkinson, Washington, D. C.; Dr. Edward Jackson, Denver, Colo.; Dr. Sanford R. Gifford, Chicago, and Dr. George P. Guibor, Chicago. Dr. Fink closed the discussion.

PRESIDENT WHEELER: The next paper is on "The Fundus Oculi in Diabetes Mellitus," by Drs. M. L. Folk and S. Soskin of Chicago.

Dr. Folk read the paper prepared by himself and Dr. Soskin.

PRESIDENT WHEELER: The discussion on Dr. Folk's paper will be opened by Dr. S. Hanford McKee of Montreal, Canada.

Dr. McKee read his discussion.

PRESIDENT WHEELER: Dr. A. D. Ruedemann, Cleveland, will continue the discussion.

Dr. A. D. Ruedemann read his discussion. Dr. Folk closed.

PRESIDENT WHEELER: The next paper is on "Social Service in Ophthalmology," by Dr. Lawrence T. Post of St. Louis and Miss Eleanor P. Brown of New York. This paper will be read by Miss Brown, who is Associate Director of the National Society for Prevention of Blindness.

MISS BROWN: Mr. President and Members: The National Society appreciates very greatly the honor of having a place on this Academy program. We, of course, know that the prevention of blindness work has the backing and coöperation of the ophthalmologists, and that gives those of us who are in the movement for the saving of sight the urge to go forward and achieve such success as may be ours.

Dr. Post and I have had some argument as to who should present this paper, with the verdict in Dr. Wheeler's hands. Dr. Post proposed the courtesy of pushing the lady forward. We

have had no argument as to the content of the paper which presents our joint point of view.

Miss Brown then read the paper prepared by herself and Dr. Post.

PRESIDENT WHEELER: Discussion of this paper will be opened by our gracious guest of honor, Professor Lauber.

Professor Lauber gave his discussion.

PRESIDENT WHEELER: Dr. Post?

DR. POST: Nothing further to add.

PRESIDENT WHEELER: Before closing this meeting, I want to thank you all very much for your coöperation. The meeting has been a very pleasant one for me and a very profitable one. I want to thank you.

Dr. Eagleton, will you come to the platform?

I have great pleasure in presenting the gavel to our new president, Dr. Eagleton.

The audience arose and applauded as President-Elect Wells P. Eagleton assumed the chair.

PRESIDENT EAGLETON: I welcome this opportunity of saying something that is very personal, because it will never occur to me again to have the opportunity to do so.

Those who have accomplished something cannot tell how great a thing they have accomplished, with delicacy, but when a man comes, as I do, into an organization and has been able to see the interior workings of it before he becomes responsible for anything, it is his privilege to tell what he sees.

I want to tell you that I was asked by Dr. Mosher some years ago to come and talk on brain abscess. I was not a member of the Academy. I said, "All right, I will prepare a paper." I have many requests like that from different parts of the country. I came and read a paper. I was seated in the back part of the hall when Dr. Gradle came and said, "Don't you think you ought to join this?"

Meanwhile, I wandered around among the instructional courses. I said, "Why, yes, I will gladly join." I joined. They assigned me a course. I did the best I could. Gradually, year after year, I gave this course, and when I wasn't giving the course I was running into other courses. I am rather an elderly

gentleman, and I found the time had come when I wished that I could have time to go to the other courses that I needed very much.

Much to my amazement, last year they elected me your president-elect, and then I was admitted to the official family; for one year I have been seated in the official family.

I want to tell you that this organization is unique in many respects, chief among which is its administration. Do you realize that this is a great big affair that moves in here, and do you realize that it is moved in by an organization under Dr. Wherry, who works all the year 'round simply for the love of working and serving the profession of which he is a member? Do you realize that that could not be bought for less than $10,000, if you paid for it, from a trained worker?

I have admiration for the administration of this society. I have been engaged in administrations with many millions of dollars. I have been a director of the second largest financial institution in New Jersey, with many millions of assets. My admiration for the administration here is without bounds.

I said yesterday, and I want to say publicly, that if President Roosevelt wanted to do something big for the health of America, he would take Dr. William P. Wherry of Omaha, Nebraska, and put him into his cabinet and turn over health matters to him, and he would operate through the doctors, and he would do a great thing for America.

The secretarial group is composed of the ophthalmological department and Dr. Gradle. There is nothing like it anywhere in the world; don't laugh; don't smile. I have seen every course of prominence in the world. That doesn't say I have taken every course, but I have visited every course in the world, from India through Africa, through North America, not South America.

The time that Dr. Benedict and Dr. Gradle give to the ophthalmological department, and the thought that they give, the success of what they are doing is phenomenal, and it is unique — two men giving up a tremendous amount of their time just simply to help the doctors who are practicing that specialty.

Now, Dr. Myers, the secretary for otolaryngology, and Dr. Mullin, secretary for instruction — if any of you have any suggestions about the program, write these men any time during the year. They will welcome it.

I want to impress this upon you: I think every man here within the sound of my voice, and every Academy member, should realize what the Academy is, and he should go to his local eye, ear, nose and throat society and tell them the story, tell them what is to be derived here. Every member who is trying to be a better eye, ear, nose and throat specialist is missing an opportunity, no matter how old he is, by not availing himself of the facilities that are here offered.

I thank you for listening, and as your president, I declare the Thirty-Ninth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology adjourned.

The meeting adjourned at twelve-ten o'clock.

THURSDAY, SEPTEMBER 13, 1934, 5:35 P.M.

Business Meeting

The Business Meeting of the American Academy of Ophthalmology and Otolaryngology, held in connection with the Thirty-Ninth Annual Session, at the Hotel Sherman, Chicago, Illinois, Thursday, September 13, 1934, convened at five-thirty-five o'clock, President Wheeler presiding.

PRESIDENT WHEELER: The business meeting will come to order.

The first order of business is the report of the Executive Secretary-Treasurer, Dr. Wherry.

EXECUTIVE SECRETARY-TREASURER WHERRY: The membership summary as of September 1, 1934, was:

Honorary Members	17	
Life Members	42	
Active Members	1,998	
		2,057
Deaths		22
Total		2,035
Candidates for Membership		115
Total names carried on the books		2,150

Members delinquent in dues total as follows:

1934	135
1933	82
1932	58
Total	275 or 13 per cent

At the 1933 Convention the delinquency percentage was 21 per cent as contrasted to 13 per cent for 1934.

The Council at this session has authorized the following changes in membership status:

Life Members — Those Fellows who have paid dues continuously for thirty years will automatically become Life Members with dues remitted.

Senior Fellows — Those Fellows who have paid dues continuously for twenty-five years will be termed Senior Fellows, and their dues will be one-half the usual amount: At the present time, five dollars annually.

Junior Fellows — All other Members (except Honorary) will be designated as Junior Members.

Honorary Memberships to:

Dr. Hans Lauber, Warsaw, Poland
Dr. Karl Koller, New York, N. Y.

Life Memberships to:

Dr. W. E. Chenery, Boston, Mass.
Dr. Oscar Dodd, Evanston, Ill.
Dr. Harry Friedenwald, Baltimore, Md.
Dr. E. B. Gleason, Philadelphia, Pa.
Dr. George W. Jean, Santa Barbara, Calif.
Dr. Edw. Meierhof, New York, N. Y.
Dr. W. H. Roberts, Pasadena, Calif.
Dr. Lillian E. Taylor, Carmel, Calif.
Dr. David W. Wells, Boston, Mass.

The following resignations have been accepted:

Dr. E. R. Bray, St. Paul, Minn.
Dr. William C. Haupt, Brooklyn, N. Y.
Dr. E. R. Lewis, Los Angeles, Calif.
Dr. Alice MacKenzie, Philadelphia, Pa.
Dr. H. M. Morton, Vincentown, N. J.
Dr. S. A. Strum, Pittsburgh, Pa.
Dr. H. A. Brown, San Francisco, Calif.

In spite of the depression, the Academy has made but little change in its general policy of operation; in fact, the program of consistent expansion has been continued.

During the current year the Teachers' Section was reorganized according to the wishes of those interested, and made an integral part of the general organization plan. Also, during the year, there was created a Division of Public Relations and Publicity. Dr. Fenton, chairman of this division, and his collaborators, Drs. King and Shea, are to be complimented upon the splendid work set up for this convention. Their services have been sincerely appreciated.

The report of the auditor is appended as a part of this report and will be published in the Transactions. Summarizing briefly, during 1934 the income per member was $15.60, deducting the cost of $14.73, leaves a surplus of 87 cents per member. The difference between this figure and the $10 dues was accounted for in miscellaneous ways.

After all disbursements, which included $3,575 given for research, there was a surplus of $1,414.53 added to the capital surplus.

The capital investment account, as of August 25, 1934, was $107,781.40, book value cost. The current market value was estimated as of $77,273.25.

On behalf of the operating officers, particularly the secretarial group, of which I am only a part, we desire to express to the President and his executive group and to the membership our sincere appreciation for the cooperation given during the current year.

Respectfully submitted,

DR. W. P. WHERRY

Executive Secretary-Treasurer

REPORT OF THE AUDITOR

Omaha, Neb., January 3, 1934

THE AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY
Omaha, Nebraska

Gentlemen:

I have examined the accounts of your Academy for the period from January 1, 1933, to December 31, 1933. I submit herewith a summary of receipts and disbursements, together with a statement of assets at December 31, 1933.

All receipts and disbursements were traced to the Academy's bank account.

The ordinary general expenses for the month of December have not been paid.

Office furniture and instruments are not listed among the assets.

The bonds owned by the Academy are in possession of the Trust Department of the Omaha National Bank for safekeeping. They were verified by a certificate from the Bank. These securities are carried at cost, with no adjustments for amortization of premium or discount.

The changes in the financial status of the Academy during the year are accounted for as follows:

Assets held January 1, 1933		$106,103.87
Add: Excess of Receipts over Disbursements		
Receipts	$28,905.45	
Disbursements	27,490.92	1,414.53
Assets December 31, 1933		$107,518.40

I certify that the statements are correct to the best of my knowledge and belief.

JOHN P. BEGLEY,
Certified Public Accountant

RECEIPTS AND DISBURSEMENTS

JANUARY 1, 1933, TO DECEMBER 31, 1933

Cash Balance, January 1, 1933		$ 322.15
Receipts:		
Applications, dues, etc.	$18,030.00	
Transactions	790.41	
Convention	6,040.04	
Bond Interest	4,045.00	28,905.45
On Capital Account:		
Bonds sold	None	
		$29,227.60
Disbursements:		
Research	$ 3,757.00	
Transactions	7,005.22	
Honorariums	500.00	
Meetings and conventions	9,781.48	
Office and general expense	6,629.22	27,490.92
On Capital Account:		
Bonds purchased	None	
		$ 1,736.68

SCHEDULE OF BONDS OWNED DECEMBER 31, 1933

	RATE	DUE	PAR VALUE	COST
Kansas City, Fort Scott and Memphis Railway, Refunding Mortgage	4%	1936	$ 5,000.00	$ 4,352.50
Chicago, Rock Is. and Pacific Ry., First and Refunding Golds Bonds	4%	1934	3,000.00	2,688.50
Midvale Steel & Ordnance Company, Convertible Sinking Fund	5%	1936	5,000.00	4,902.64
Denver Gas & Electric Company, General Mortgage	5%	1949	4,000.00	3,951.00
Witherbee Sherman Company, First Mortgage Sinking Fund Series "A"	6%	1944	5,000.00	4,925.00
Commonwealth Edison Company, First Mortgage and Collateral Trust Series "C"	4½%	1956	5,000.00	4,825.00
Detroit City Gas Company, First Mortgage Gold Bonds Series "B"	5%	1950	5,000.00	5,025.00
Kingdom of Denmark, Gold Bonds	5½%	1955	3,000.00	3,030.00
Youngstown Sheet & Tube Company, First Mortgage Series "A"	5%	1978	5,000.00	5,003.75
Columbia Gas & Electric Company, Debenture Bonds	5%	1952	5,000.00	4,997.50
Van Sweringen Company, First Mtg. and Coll. Trust, Sinking Fund Gold Bonds	6%	1935	6,000.00	6,031.35
Missouri Pacific Railroad, First and Refunding Mortgage	5%	1978	2,000.00	1,934.00
Chicago, Milw., St. Paul & Pacific, First Mortgage Gold Bonds Series "A"	5%	1975	2,000.00	1,821.50
Goodyear Tire and Rubber Company, First Mortgage and Collateral Trust Bond	5%	1957	2,000.00	1,839.00
American International	5½%	1949	2,000.00	1,962.50
Union Gulf	5%	1950	2,000.00	2,030.00
Massachusetts Gas Company	5%	1955	2,000.00	1,970.00
Kingdom of Norway	5%	1963	2,000.00	1,995.00
Central Illinois Public Service, First Mortgage Gold Bonds	5%	1968	3,000.00	2,925.00
Citizens Bldg. Co. of Cleveland, 1st Mtg. Sink. Fund Gold Bds. and 50 shares stock	6%	1946	5,000.00	5,000.00
Inland Steel Co., Series "B" Gold Bonds	4½%	1981	5,000.00	4,825.00
Interlake Iron Corp., Gold Bonds Series "B"	5%	1951	5,000.00	4,850.00
Missouri Pacific Railroad Company, First Mortgage Refunding Series 1	5%	1981	3,000.00	2,850.00
Public Service Co. No. Ill., First and Refunding	4½%	1981	5,000.00	4,875.00
Southern California Gas Co., First Mortgage and Refunding Gold	4½%	1961	5,000.00	4,775.00
Southern Pacific Company	4½%	1981	5,000.00	4,837.50
Brooklyn Edison Co., General Mortgage Gold Bond Series "E"	5%	1952	3,000.00	2,910.00
Columbus Railway Power & Light Company, Convertible Gold Bonds	5½%	1942	2,000.00	1,880.00
Dominion of Canada, War Loan 20 Year Gold Bonds	5%	1937	3,000.00	2,790.00
Ophthalmic Publishing Co., Stock, Five Shares			100.00	Gift
			$109,100.00	$105,781.72

SUMMARY OF ASSETS, DECEMBER 31, 1933

Cash Balance	$ 1,736.68
Securities owned (at cost)	105,781.72
	$107,518.40

BANK RECONCILEMENT

OMAHA NATIONAL BANK, OMAHA, NEBRASKA

Balance per Bank Statement, December 31, 1933	$1,736.68

No checks were outstanding.

PRESIDENT WHEELER: We will next hear from Dr. Babbitt, the historian.

DR. JAMES A. BABBITT: Mr. President and Gentlemen of the Academy: The historian has already made a report to the Council and prepared a somewhat shorter list of memorial notices for the men who have died this past year, and is of the opinion that the former members of the society may be more elaborately commented upon in the proceedings. It seems best now to simply read the list of those who have left us this past year.

In Memoriam

Dr. Harvey Mayor Becker, Sunbury, Pennsylvania
Dr. William Ben Boyce, Escanaba, Michigan
Dr. Lizzie Maud Carvill, Boston, Massachusetts
Dr. Jeremiah Joseph Corbett, Boston, Massachusetts
Dr. Josiah Wellington Crane, Trenton, New Jersey
Dr. Ira Oscar Denman, Toledo, Ohio
Dr. John Forest Dickson, Berkeley, California
Dr. Albert Marion Earel, Hoopeston, Illinois
Dr. Thomas Faith, Chicago, Illinois
Dr. William Chris Finnoff, Denver, Colorado
Dr. Frederick Erasmus Franchere, Sioux City, Iowa
Dr. James Cole Hancock, Brooklyn, New York
Dr. George Paul Marquis, Chicago, Illinois
Dr. John Sabert Mott, Kansas City, Missouri
Dr. Charles Aloysius O'Reilly, Devon, Pennsylvania
Dr. Edward Mahlon Palmer, Wichita, Kansas
Dr. William Campbell Posey, Philadelphia, Pennsylvania
Dr. Charles Richard Reaves, Greensboro, North Carolina
Dr. Monroe David Reese, Lebanon, Pennsylvania
Dr. Maximilian Adolph Schurter, Long Beach, Calif.
Dr. Dero Eugene Seay, Dallas, Texas
Dr. Eustace Monett Singleton, Marshalltown, Iowa
Dr. Raymond John Sisson, Detroit, Michigan

The following memorial notices were submitted by the historian.

Dr. Harvey Mayor Becker

Dr. Harvey Mayor Becker was born in Hanover, Pennsylvania, on May 3, 1873, and died in Sunbury, Pennsylvania, on August 28, 1933.

Dr. Becker received his preliminary education in the public schools of Hanover, prepared at Gettysburg College and received his medical degree from the University of Pennsylvania, followed by postgraduate work at the Philadelphia Polyclinic and the Wills Eye Hospitals in Philadelphia, the New York Polyclinic Hospital, the New York Eye and Ear Infirmary and the New York Eye, Ear and Throat Surgery Hospital.

In 1898, immediately after his graduation, Dr. Becker located in Sunbury. He was superintendent of the Mary M. Packer Hospital for many years, holding that position up until the time of his fatal illness. He was also a member of the surgical staff of the Geisinger Hospital at Danville for a number of years.

He was a member of the Northumberland County Medical Society, the Pennsylvania State Medical Society, American Medical Association, Philadelphia Laryngological Association and the American Academy of Ophthalmology and Otolaryngology.

Dr. Becker is survived by his wife and one daughter, Miss Anna Louise Becker.

Dr. William Ben Boyce

Dr. William Ben Boyce was born in Dartford, Ontario, Canada, on February 21, 1874, and died in Escanaba, Michigan, December 31, 1933.

Dr. Boyce received his degree in medicine at the University of Toronto and, following his graduation, came to the United States, starting his general medical practice at Bark River in 1902. A few years later he went to Chicago to take special work in the treatment of diseases of the ear, nose, throat and eye, and, upon the completion of his course, opened his office in Escanaba, continuing his practice up to the time of his fatal illness.

He was a member and former president of the Delta County Medical Society, a member of the Michigan State Medical Society and the American Academy of Ophthalmology and Otolaryngology, and on the staff of St. Francis Hospital.

Dr. Boyce is survived by his wife and four children, three brothers and two sisters.

Dr. Lizzie Maud Carvill

Dr. Lizzie Maud Carvill was born in Lewiston, Maine, daughter of Dr. Alphonso H. Carvill, and died February 25, 1934, in Somerville, Massachusetts, at the age of sixty years.

Dr. Carvill received her preliminary education in the public schools of Somerville and the School of Physical Training, Cambridge. In 1899, she received her B.A. degree from Tufts College, her medical degree from Tufts Medical Society in 1905, and took postgraduate studies at Harvard Medical School, specializing in eye, ear, nose and throat work.

She became an assistant surgeon in ophthalmology at the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital, retiring from the latter about a year before her death. She served also as visiting ophthalmologist to the New England Hospital for Women and Children for over twenty years, retiring in 1929 to become a consultant.

Dr. Carvill was a member of the New England Women's Medical Society, the Massachusetts Medical Society, the American Medical Association, the New England Ophthalmological Society, the American College of Surgeons, the American Academy of Ophthalmology and Otolaryngology and the American Ophthalmological Society. Of the last named society, she was the only woman member winning admission by a thesis on glaucoma. She is cited as one of the American authorities on interstitial keratitis and tuberculosis of the eye. At the International Congress of Ophthalmologists in Washington in 1922 she presented a paper "Bitemporal Contraction of the Visual Fields in Pregnancy." Among other articles from her pen are "A Contribution to the Study of Phlyctenula-Keratitis" and "Persistent Hyaloid Artery."

Dr. Carvill is survived by a brother, Sewall Albert Carvill of Somerville, Massachusets, two nephews, a grandnephew and a grandneice.

Dr. Jeremiah Joseph Corbett

Dr. Jeremiah Joseph Corbett was born in Peabody, Massachusetts, on August 28, 1877, and died in Santa Fe, New Mexico, on August 24, 1933.

Dr. Corbett received his early education in the Peabody public schools, and took preparatory work at St. Francis Xavier College at Antigonish, Nova Scotia, Canada, receiving his medical degree from Harvard College in 1906. After serving his interneship at Boston City Hospital, he specialized in eye, ear, nose and throat work, and located in Malden, Massachusetts. In addition, he made many trips to Europe and studied in the clinics of Paris, Vienna and other medical centers in the interests of his chosen speciality.

For ten years Dr. Corbett was head of the eye department of the Boston City Hospital, and head of the eye, ear, nose and throat department of the Malden Hospital. In addition, he was on the staff of the Carney Hospital, St. Elizabeth's Hospital, Boston, Anna Jacques Hospital, Newburyport, and the Whidden Memorial Hospital.

He was a member of the New England Ophthalmological Society, the New England Otological and Laryngological Society, the American College of Surgeons and the American Academy of Ophthalmology and Otolaryngology.

Dr. Corbett was interested in all civic matters and held membership in many local social societies.

He is survived by his wife and one son, Francis Xavier Corbett.

Dr. Josiah Wellington Crane

Dr. Josiah Wellington Crane died February 18, 1934, at Rochester, Minnesota, at the age of fifty-six years.

Dr. Crane received his medical degree from the University of the South Medical Department, Sewanee, Tennessee, in 1900.

He was a member of the Medical Society of New Jersey, American Academy of Ophthalmology and Otolaryngology.

For a number of years he had served as Medical Director of the New Jersey State Prison Hospital.

Dr. Ira Oscar Denman

Dr. Ira Oscar Denman was born in Leanna, Kansas, on June 9, 1872, and died in Toledo, Ohio, September 28, 1934.

Dr. Denman attended the Allan County Normal School in Iola, Kansas, and received his medical degree from Hahnemann Medical College in Chicago in 1897, following which he did post-graduate work in the Medical College, New York Eye and Ear Infirmary, Harvard Medical College, the Universities of Vienna and Frieberg, and in Moorfield's Hospital, London, England. He began his practice in general medicine and surgery of the eye, ear, nose and throat in Charleston, Illinois, and in January, 1908, moved to Toledo, Ohio, to limit his practice to eye, ear, nose and throat work.

He became chief of staff of the Toledo Hospital in 1913, and was also oculist for the Pennsylvania Railroad, the Detroit and Toledo Shore Line Railroad, Toledo, St. Louis and Western Railway, and Chairman of Board of Health in Charleston, Illinois, from 1903 to 1907.

His memberships included the American Board of Otolaryngology and American College of Physical Therapy, American Medical Association, the American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, the American Institute of Homeopathy, Illinois State Medical Society, Ohio State Medical Society, Northwestern Ohio Medical Society, Lucas County Academy of Medicine. He was on the editorial staff of the Archives of Physical Therapy, X-ray and Radium, and co-inventor of the vocaphone and artificial larynx for use in talking after a laryngectomy.

Dr. Denman is survived by his wife, two daughters and one son.

Dr. John Forest Dickson

Dr. John Forest Dickson was born in Toronto, Canada, in 1856, and died at Berkeley, California, on April 3, 1934.

Dr. Dickson graduated from the University of Toronto Faculty of Medicine in 1880, and from Victoria Medical College, following which he went to the University of Edinburgh, Scotland, graduating with honors in 1889. He began his special work at the Moorfield's Eye Hospital and the Grey's Inn Road Ear, Nose and Throat Hospital, London, later visiting the special clinics in Paris and Berlin.

In May, 1891, Dr. Dickson began his practice of ophthalmology and otolaryngology in Portland, Oregon, which he continued up until 1931, becoming Professor of Ophthalmology at the Uni-

versity of Oregon Medical School, and making many trips to New York, Boston and Philadelphia clinics, as well as to Vienna, where he worked with Dr. Ernest Fuchs. At the time of his death Dr. Dickson was emeritus professor of ophthalmology at the University of Oregon Medical School.

He was a member of the Oregon State Medical Society, the Pacific Coast Oto-Ophthalmological Society, the American College of Surgeons, American Laryngological, Rhinological and Otological Society, and the American Academy of Ophthalmology and Otolaryngology.

Dr. Dickson is survived by his wife.

Dr. Albert Marion Earel

Dr. Albert Marion Earel was born June 22, 1866, in Abingdon, Illinois, and died in Hoopeston, Illinois, on February 25, 1934.

He received his preliminary education in the district schools and Knox College, embarking on his medical studies under the preceptorship of Dr. Madison Reece of Abingdon, and received his degree of M.D. at Rush Medical College in 1891. He did postgraduate work at the Chicago Eye, Ear, Nose and Throat College, the Philadelphia Polyclinic Hospital, Wills Eye Hospital, Philadelphia, Central Eye and Ear Infirmary and Polyclinic of New York City, and the Golden Square Nose and Throat Hospital and Guy's Hospital in London, England, also studying in Vienna.

Dr. Earel was a member of the Vermilion County Medical Society, the American Medical Association and the American Academy of Ophthalmology and Otolaryngology.

Dr. Earel is survived by his wife and one daughter.

Dr. Thomas Faith

Dr. Thomas Faith was born in Hancock, Maryland, and died in Chicago, Illinois, October 16, 1933, at the age of sixty-one years.

His family moved to Warrensburg, Illinois, in his childhood, and he received his early education in the schools of Decatur, Illinois, and Rush Medical College, and graduated from the College of Physicians and Surgeons of Chicago in 1893.

For many years Dr. Faith was instructor in ophthalmology at the Chicago Eye, Ear and Throat College, and was on the

staff of the South Shore Hospital. He was a former president of the Chicago Ophthalmological Society.

Dr. Faith was a member of the Illinois State Medical Society, the College of Physicians and Surgeons of Chicago, the Chicago Ophthalmological Society and the American Academy of Ophthalmology and Otolaryngology.

Dr. Faith is survived by his wife and one son.

Dr. William Chris Finnoff

Dr. William Chris Finnoff was born in Pittsburgh, Pennsylvania, on June 25, 1890, moved to Denver, Colorado, in 1903, and died there on June 10, 1934, of heart disease.

Receiving his preliminary education in the Denver schools, he graduated in medicine from the University of Colorado in 1912, and received the degree of Doctor of Ophthalmology from the same institution in 1915, practicing his specialty in Denver. During the World War he served in France and England, with the rank of Captain in the Medical Corps, and studied at the Royal London Ophthalmic Hospital (Moorfield's), while stationed in London.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society and the American Medical Association, and was Associate Professor of Ophthalmology in the University of Colorado.

Dr. Finnoff made many contributions of important papers to the literature of ophthalmology, nearly half of which dealt with ocular tuberculosis.

He is survived by his widow and three children.

Dr. Frederick Erasmus Franchere

Dr. Frederick Erasmus Franchere was born in North San Juan, California, on July 14, 1867, and died in Sioux City, Iowa, on April 28, 1934.

Dr. Franchere graduated from the University of Minnesota with the degrees of B.S. and M.D., and interned in the Minnesota State Hospital in Rochester, followed by a year's study in Paris under Charcot.

He was a member of the Iowa State Medical Society, past president of the Woodbury County Medical Society, Sioux City

Eye and Ear Society, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology and Gorgas Memorial Society. He was formerly professor of nervous diseases, secretary of the faculty and professor of ophthalmology and otolaryngology at the Sioux City College of Medicine. At one time Dr. Franchere served as coroner of Watonwan County, Minnesota, and health officer of St. James, Minnesota. At the time of his death, Dr. Franchere was on the staffs of St. Vincent's Hospital and the Lutheran Hospital.

In addition to his medical work, Dr. Franchere served on the Executive Council of the Boy Scouts, was a member and past president of the Sioux City chapter of the American Interprofessional Institute, was for five years editor of The Bystander, a department of the Institute's national magazine devoted to book notes and art, was secretary of the Sioux City Fine Arts Society and a member of the Independent Artists of America and of the Iowa Artists' Association.

Dr. Franchere is survived by his wife, two daughters, one son and a granddaughter.

Dr. James Cole Hancock

Dr. James Cole Hancock was born in Fulton, N. Y., on April 24, 1865, and died at his home in Brooklyn, N. Y., on November 27, 1933.

His early education was received in the public schools and Polytechnic Institute of Brooklyn, and his medical degree obtained from the College of Physicians and Surgeons of Columbia University in 1889. Following his graduation he served as resident surgeon in the Chambers Street Hospital and the New York Eye and Ear Infirmary, and then went to Berlin, Paris and London to perfect his studies in Ophthalmology. On his return he took up the practice of his specialty in Brooklyn.

His positions included being an assistant surgeon in the New York Eye and Ear Infirmary and surgeon in the old Eastern District Hospital of Brooklyn, and consulting ophthalmologist to the Long Island State and Coney Island Hospitals of Brooklyn and the Hempstead Hospital of Hempstead, Long Island.

Dr. Hancock was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and the American Medical Association, and a member of the Medical Society of the County of Kings, Medical Society

of the State of New York, the Brooklyn Ophthalmological and Brooklyn Neurological Societies and the Associated Physicians of Long Island.

Dr. Hancock was the author of many papers on ophthalmological subjects.

Dr. George Paul Marquis

Dr. George Paul Marquis was born in Pittsburgh, Pennsylvania, September 12, 1868, and died in Chicago on December 22, 1933.

Dr. Marquis attended the public schools in Chicago and graduated from Washington and Jefferson University with the degree of A.B. in 1889, receiving his A.M. from the same university in 1892, and his medical degree from Northwestern University Medical School in 1892. He served his interneship in Chicago, and took postgraduate studies in Berlin, Vienna and Freiburg from 1895 to 1897, going abroad again for study on several later occasions.

He was assistant professor of laryngology and rhinology at Northwestern University Medical School, attending otolaryngologist to St. Luke's Hospital and a veteran of the Spanish-American War.

Dr. Marquis was a member of Phi Kappa Psi Fraternity, the American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Society, the Chicago Institute of Medicine, Chicago Laryngological Society, Chicago and Illinois State Medical Societies, American Medical Association and the American College of Surgeons.

Dr. John Sabert Mott

Dr. John Sabert Mott was born in St. Marys, Ohio, and died in Kansas City, Missouri, on January 7, 1934, at the age of eighty-nine years.

Dr. Mott attended West Point Military Academy for two years and left there in 1864 to join the 180th Ohio Volunteers as a Second Lieutenant in the Civil War. In 1865, he was appointed First Lieutenant of the 118th Ohio Volunteers. He was a member of the original Military Order of the Loyal Legion of the United States, composed of officers of the Revolutionary and Civil Wars.

Dr. Mott was a member of the American Academy of Ophthalmology and Otolaryngology and an affiliate Fellow of the American Medical Association.

He is survived by his wife, two daughters, two sons and two grandchildren.

Dr. Charles Aloysius O'Reilly

Dr. Charles Aloysius O'Reilly died in Devon, Pennsylvania, on November 1, 1933, at the age of fifty-five years.

He received his medical degree from the University of Pennsylvania School of Medicine, Philadelphia, in 1900, and served during the World War.

Dr. Edward Mahlon Palmer

Dr. Edward Mahlon Palmer was born in Chillicothe, Missouri, on June 18, 1874, and died in Wichita, Kansas, on December 31, 1933.

Dr. Palmer graduated from the University Medical School at Kansas City, now the University of Kansas, in 1904, and served in the Spanish-American War as First Sergeant in Company H, 4th Missouri Volunteers.

He was past president of Wesley Hospital staff, and a charter member of the staff.

He was a member of the Sedgwick County Medical Society, Kansas Medical Society, American Medical Association, American College of Surgeons and the American Academy of Ophthalmology and Otolaryngology.

Dr. Palmer is survived by his wife, one son and one daughter, three sisters and two brothers.

Dr. William Campbell Posey

Dr. William Campbell Posey, of Radnor, Pennsylvania, was born in Philadelphia on July 5, 1866, and died on board the Steamship Rex, in the harbor at Naples, Italy, on September 5, 1934.

His early education was gained in the Episcopal Academy, and he received the A.B. degree from the University of Pennsylvania in 1886, graduating with the degree of M.D. in 1889. After serving his interneship in the Presbyterian Hospital, Phila-

delphia, Dr. Posey studied ophthalmology in Berlin, Goettingen, London, and Paris, returning to Philadelphia in 1892 to take up the practice of ophthalmology. He had retired from active practice several years ago.

Dr. Posey was professor of ophthalmology in the Philadelphia Polyclinic Hospital and for many years surgeon to the Wills Eye Hospital, the Howard Hospital and the Children's Hospital. A member of his county and state medical societies, he was the first chairman of the Commission on Conservation of Vision appointed by his state society. He was Fellow of the College of Physicians of Philadelphia, being president at the time of his death, a Fellow of the American Medical Association and Chairman of the Section on Ophthalmology in 1910, and a Fellow of the American College of Surgeons. He was a member of the American Ophthalmological Society, serving as president in 1929, a member of the American Academy of Ophthalmology and Otolaryngology, and the American Board of Ophthalmology. He was also a member of several clubs, the Phi Kappa Psi fraternity and the Sons of the American Revolution.

Dr. Posey edited the sixth American edition of Nettleship's Diseases of the Eye, was author of a book on Hygiene of the Eye, co-author of a treatise on Diseases of the Eye, Nose, Throat and Ear, and contributed extensively to ophthalmic literature.

He is survived by his widow and two daughters.

Dr. Charles Richard Reaves

Dr. Charles Richard Reaves was born in Greenville, Tennessee, and died in Greensboro, North Carolina, on February 6, 1934, at the age of forty-nine years.

He graduated from Greenville High School and the University of the South at Sewanee, Tennessee, getting his medical degree from the University of the South in 1907, afterward doing postgraduate work in the clinics of Chicago and New York, specializing in eye, ear, nose and throat.

He was a member of the North Carolina State Medical Society, the Medical Arts Club, the American Medical Association, American Academy of Ophthalmology and Otolaryngology and vice-president and program chairman of the Guilford County Medical Society. He was on the staff of St. Leo's Hospital.

Dr. Reaves is survived by his wife and children.

Dr. Monroe David Reese

Dr. Monroe David Reese was born in Ashland, Pennsylvania, on March 31, 1897, and died in Lebanon, Pennsylvania, on March 22, 1934.

In his youth he attended the public schools of Ashland, and graduated from the University of Pennsylvania in June, 1921, receiving his medical degree from that University in 1925. Following his graduation he was associated for some time in the office of Dr. George M. Coates of Philadelphia, taking up his practice in eye, ear, nose and throat work in Lebanon, Pennsylvania, in 1926, being associated with Dr. F. S. Kauffman for ten months and taking over his office and private hospital when Dr. Kauffman died.

He was a member of the John B. Deaver Surgical Society, the Ptolemy Society of the University of Pennsylvania, the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, and past president of the Lebanon County Medical Society. While a student at the University of Pennsylvania, Dr. Reese served in the Students' Army Training Corps.

Dr. Reese is survived by his wife.

Dr. Maximilian Adolph Schurter

Dr. Maximilian Adolph Schurter was born in Zurich, Switzerland, on March 2, 1882, and died in Long Beach, California, on August 22, 1933.

He was graduated from the medical school of Long Island College, New York City, in 1912, and after serving his internship went as a medical missionary to Porto Rico, representing the Congregational Board of Missions. For ten years he helped build the Ryder Memorial Hospital, the largest in Porto Rico, and was head of the hospital.

Returning to the United States after the World War, Dr. Schurter took a four-year advanced course in ophthalmology and otolaryngology at the University of Pennsylvania, Philadelphia, and moved with his family to Long Beach to begin practice of his specialties in 1923.

Dr. Schurter was on the staff of the Seaside Hospital, a member of the local and Los Angeles County Medical Associations, and of the American Academy of Ophthalmology and Otolaryngology.

He is survived by his wife, three sons and three daughters.

Dr. Dero Eugene Seay

Dr. Dero Eugene Seay died at his home, 3421 Beverly Drive, Dallas, Texas, on November 16, 1933, aged 59 years.

Graduating in medicine from Vanderbilt University and serving his interneship there and at Bayvue Hospital in Baltimore, Dr. Seay took up the practice of his specialty in Dallas.

Dr. Seay was a member of the American Medical Association, Texas State Medical Association, Dallas County Medical Society, American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, Texas Eye, Ear, Nose and Throat Society, and was on the staff of St. Paul's Hospital in Dallas.

A widow and three sons survive him.

Dr. Eustace Monett Singleton

Dr. Eustace Monett Singleton was born in Eyota, Minnesota, on January 17, 1864, and died in Marshalltown, Iowa, on November 29, 1933. Dr. Singleton's early education was gained from the Olmsted County Schools, and his medical degree from Chicago Medical College in 1891. After hospital and postgraduate studies, he entered private practice in his specialty of eye, ear, nose and throat work in 1894 in Marshalltown, Iowa. In 1902, he started the first hospital in Marshalltown and conducted a Training School for Nurses.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and an indefatigable worker for the advancement of his specialty.

His widow and one daughter survive him.

Dr. Raymond John Sisson

Dr. Raymond John Sisson was born in Syracuse, New York, on May 28, 1897, and died in Boston on February 23, 1934.

Dr. Sisson graduated in medicine from the University of Syracuse in 1922, serving his interneship in Henry Ford Hospital, and became Resident in Medicine at Lenox Hill Hospital, New York, afterward taking up practice in his specialty in Detroit, Michigan.

He served during the World War and was on the staffs of the Harper Hospital, Children's Hospital of Michigan and St. Mary's Hospital, all in Detroit.

Dr. Sisson was the author of several medical papers and was a member of the American Medical Association, American College of Surgeons and the American Academy of Ophthalmology and Otolaryngology.

He is survived by his wife, his mother and one brother.

DR. BABBITT: I think it would be appropriate for our Fellowship to stand for a moment in tribute to those who have left us this past year.

Silent tribute to deceased members.

PRESIDENT WHEELER: Dr. Beach will read the report of the American Board of Ophthalmic Examinations.

REPORT OF THE AMERICAN BOARD OF OPHTHALMOLOGY

DR. S. JUDD BEACH: Mr. President and Members: It is proper to call attention to the change of name of this Board from American Board of Ophthalmic Examinations to the American Board of Ophthalmology, making the name uniform with that of the other specialty boards.

The American Board of Ophthalmology has conducted to date 45 examinations and has certificated 1,164 ophthalmologists.

Examinations held during 1934:

Cleveland, June 11, 1934 ..... 23 candidates
Butte, July 17, 1934 ..... 16 candidates
San Francisco, July 24, 1934 ..... 16 candidates
Chicago, September 8, 1934 ..... 34 candidates

Contemplated examinations, 1934-35:

San Antonio ..... November 13, 1934
Philadelphia and/or New York ..... June 8, 1935

The change in methods of examination and increase in work of the Board has made it desirable to enlarge the membership. For these reasons, and to insure larger attendance at examinations, the Board, on June 11, 1934, voted to request the three component societies represented on the Board to elect a fourth

representative to membership; that the term of representation be four years, and that no member of the Board may serve consecutively more than one full term. It further requests that the terms of the present representatives be increased by one year so as to make the terms four years instead of three years.

The American Board of Ophthalmology has joined with the other special medical boards to form the Advisory Board for Medical Specialties. The following special boards are also represented:

American Board of Otolaryngology
American Board of Obstetrics and Gynecology
American Board of Dermatology and Syphilology

In addition to these special boards, the following organizations are included in this Advisory Board for Medical Specialties:

Association of American Medical Colleges
National Board of Medical Examiners
Federation of State Medical Boards of the U. S. A.
American Hospital Association
Council on Medical Education and Hospitals,
American Medical Association

The function of this Advisory Board is to coordinate the efforts of the individual boards in bringing about special courses at the universities and medical colleges for the training of medical specialists.

The American Board of Ophthalmology is represented by Drs. Lancaster and Wilder, who have attended the meetings devoted to the organization of the Advisory Board. The first meeting of this Advisory Board was held at Cleveland on June 10, 1934.

This organization is a recognition of the increasing importance of the work of the specialty examining boards instituted on the pattern inaugurated by this Board in 1917. Others now forming are expected to join.

Representation of the American Academy of Ophthalmology and Otolaryngology on the American Board of Ophthalmology:

Dr. F. P. Calhoun.....1st term ends Dec. 31, 1934
Dr. S. Judd Beach.....2nd term ends Dec. 31, 1935
Dr. Wm. H. Wilder...2nd term ends Dec. 31, 1936

Officers of the Board for 1935:

President .....Dr. Luther C. Peter
Vice-President.....Dr. John Green
Secretary-Treasurer.....Dr. William H. Wilder
Assistant Secretary.....Dr. Thomas D. Allen

PRESIDENT WHEELER: I am going to call on Dr. McKee, Senior Member of the Council, to give his report at this time.

REPORT OF SENIOR MEMBER OF COUNCIL

DR. S. HANFORD MCKEE: Mr. President and Members: The Council begs to report as follows:

Serious discussion was had upon the convention setup, especially the placement of the golf party. The Council has decided to place this activity on Friday of the convention period.

The Transactions for 1934 are to be dedicated to Dr. H. P. Mosher of Boston.

An engraved certificate of membership will be given to newly elected Fellows. Present members may secure a certificate at a moderate cost. *This arrangement will become effective after the 1935 meeting.*

The Guild Craft of Optometrists has asked the Academy to assist in financing an advertising program. It was thought unwise to participate.

Because of the increasing number of activities, and because it is a fixed policy that each activity be under the direction of a secretary, the Council has added a new member to the secretarial group, with the title of Secretary for Miscellaneous Activities.

The Council, feeling that every effort should be made to assist new members in starting their Transactions file, has authorized the following schedule:

To members elected during the past two years:

Volumes recording the 1932 and 1933 meetings may be purchased at $2.00 each.

Volumes of the years prior to 1932 — $1.00 each.

To members elected previous to 1933:

Copies of past meetings may be purchased for $2.00 each.

Research appropriations for 1935 have been made as follows:

Dr. McNally, Montreal	$750.00
Dr. Larsell and Dr. Fenton, Portland, Ore.	250.00
Dr. Lurie, Boston	200.00
Committee on Optics and Visual Physiology	200.00
Committee on Trachoma	100.00
Army Medical Museum	500.00
Dr. Fry, Washington University	500.00

Respectfully submitted,

DR. S. HANFORD MCKEE,
Senior Member of Council

Upon motion, regularly made and seconded, the report of the Senior Member of the Council was accepted.

PRESIDENT WHEELER: For the American Board of Otolaryngology, Dr. Mosher will report.

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

DR. H. P. MOSHER: Mr. Chairman and Members of the Society: The American Board has functioned according to schedule for the past year and has functioned actively. Three examinations have been held, including this one; the first one at Cleveland, where about seventy were examined; a special examination in Butte for the men of the Pacific Coast, those who could not get fully East, where there were fourteen, and a skeleton board acted; and the examination here, where sixty-two men of the seventy who applied were examined. Seventeen examiners were used in this last examination. We want to thank the Chicago men, Dr. Gradle especially, for furnishing the facilities for the examination.

Dr. Beach has spoken about the Advisory Council, which has a very strong political background, which we hope is not endowed with some political aspirations of certain of the societies — to be specific, the American Medical. It is an Advisory Council by its constitution, and, if constitutions mean anything now, it will be kept as an Advisory Council. So much for the report.

Thank you, gentlemen, for the very signal honor of dedicating the Transactions to me.

Reports of Standing Committees

PRESIDENT WHEELER: Dr. Benedict will report for the American Committee on Optics and Visual Physiology.

AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

DR. WILLIAM L. BENEDICT: The American Committee on Optics and Visual Physiology is composed of three members from each of the national societies, three members from this group.

The studies on visual acuity are still being carried out in the Department of Psychology of the University of Michigan.

The following foreign instruments have been found useful for the orthoptic treatment of heterophoria and heterotropia: the Synoptiscope (Clement Clark, London), the Synoptophore (Curry and Paxton, London), and the Orthoptiscope (Theo. Hamblin, London). Serious consideration should be given to the simplification and reduction of costs of these instruments.

The Committee recommends that visual acuity of 20/20 without correcting lenses in each eye, be required as the minimum visual standard for admission to the United States Military Academy.

The Committee discussed and approved the suggestion that all advertising matter concerning optical apparatus, including lenses, be referred to and approved by this Committee, before publication in the American Journals of Ophthalmology.

The syllabus on Optics and Visual Physiology adopted by the American Board of Ophthalmology was approved as the minimum requirement.

Respectfully submitted,

C. S. BAHN
S. J. BEACH
WM. L. BENEDICT
A. COWAN
J. N. EVANS
W. B. LANCASTER
W. H. LUEDDE
C. BERENS, *Secretary*
E. JACKSON, *Chairman*

PRESIDENT WHEELER: Committee on Extramural Postgraduate Instruction, Dr. Mullin.

EXTRAMURAL POSTGRADUATE INSTRUCTION

DR. W. V. MULLIN: Mr. Chairman, your Committee has taken advantage of our simultaneous attendance at the national meetings and have held meetings at that time, and, in addition to that, in this past year have held one special meeting of a day's work here in Chicago.

We can report progress in the past year, but the Committee feels that its true objective is far from attainment. It still is of the opinion that there is need for more and properly arranged postgraduate instruction throughout the country.

We feel that we are in a position to be of real help to any society, group or school promoting a postgraduate course.

It is our desire and wish that all postgraduate courses would be listed with this Committee, that we might give out information as to the curriculum, the time and the tuition of the course.

There have been two postgraduate courses given with the cooperation of this Committee since the last annual meeting of this society.

We would again like to emphasize that it is not the desire of this Committee to dictate nor manage any postgraduate course, but rather to stimulate and encourage more postgraduate instruction, and to cooperate in a helpful way with those doing it.

I might add further that we have on file a complete cross-section of instructors, of subjects, of the time of the year it would be possible to obtain the instructors to participate in any postgraduate instruction course, so that I again reiterate that we feel that we could be of help to anyone who desires to put on such a course.

I might say that during this meeting we have had several requests, and I hope that we will be able to help considerably those who have made such requests from us.

W. V. MULLIN, *Chairman*
WM. L. BENEDICT
H. S. GRADLE

PRESIDENT WHEELER: For the National Otolaryngological Library, Dr. Beck.

NATIONAL OTOLARYNGOLOGICAL LIBRARY

DR. JOSEPH C. BECK: Mr. Chairman, this Committee has had under discussion a general plan for the organization of a central library for ear, nose and throat. The five component societies, including the Section of the American Medical Association, have each appointed a member of the Committee.

Unusual depression has held in abeyance the original outline of a plan to establish a library in some central city of the Mississippi Valley, to be owned and operated, and controlled by these five component societies, has been found impractical on account of the finances and difficulty of raising a sufficient amount of money to establish and maintain such a library.

We have been in conference with the Surgeon General's Library from time to time and we have joined with the A. M. A. in bringing before Congress a special request for an appropriation for a new library building to properly house the magnificent library of the Surgeon General's office, the largest in the world, and to allow for expansion and provide for special provision for the libraries, including reprints and volumes in all languages, indexed and properly assorted and assigned to a definite portion of the new building. At present, the old Surgeon General's library is too small to segregate the volumes and literature of all specialties. We hope in the near future to accomplish this purpose. We ask special and individual cooperation and influence be addressed to each Congressman and Senator that they may recognize and understand the importance of the new building to the entire medical profession.

In communication with Major Hume, Chief of the Surgeon General's Library, this is what he said: "It is impossible at the present time to allow more space for the expansion of these ideas. We anticipate that, within a few years, this will be workable. In the meantime, the Index Medicus and the extension service of the Surgeon General's Library is available to all members of this Society." The Research Committee of this Academy has allotted $500 to the library for the collection and use of such pathologic specimens and volumes as may be desired from time to time, and communication with the Surgeon General's Library will furnish each member with the valuable books and specimens that may be advantageous to those preparing special papers or theses.

PRESIDENT WHEELER: For the Committee on Prevention and Amelioration of Deafness, Dr. Newhart.

PREVENTION AND AMELIORATION OF DEAFNESS

DR. HORACE NEWHART: Your Committee is pleased to report for the past year greater activities of wider scope, made possible through the grant of funds by the Council at the Boston meeting.

In this connection, Mr. Chairman, we wish to express our appreciation and thanks for the further grant just mentioned in Dr. McKee's report. It will be put to good use.

The need and value of the periodic audiometric testing of school children as an important feature of the school health program has been effectively presented in seven teachers' colleges. For their educational value, surveys of the hearing of the faculties, student bodies, children in the training schools and public schools were made, and lectures were given before medical and parent-teacher groups. By request, similar programs were carried out in other cities with a resultant demand that the surveys be regularly continued by the school authorities.

To help overcome the very obvious lack of knowledge concerning the possible accomplishments in the line of deafness prevention and the conservation of hearing, your Committee prepared a scientific, education exhibit for the meeting of the Minnesota Medical Association at Duluth in August, 1934. This proved so successful in awakening interest both among physicians and the public that on the request of the Minnesota Public Health Association, this exhibit was repeated on a larger scale at the Minnesota State Fair in September. The result was most gratifying. A large amount of carefully selected literature was distributed among sincerely interested inquirers. The dissemination of this knowledge will increase the demand for the services of physicians, especially otolaryngologists.

These exhibits, as well as other activities, have been made possible by the generous cooperation of various national, state and local organizations. Expensive equipment has been loaned by the Bell Telephone Laboratories, the American Federation of Organizations for the Hard of Hearing, the University of Minnesota Medical School and the Minneapolis public schools.

The work during the past year has included several investigations and research projects along lines fundamentally related

to activities of this Committee. They involve studies among the inmates of several Minnesota public institutions, including the School for the Deaf, the School for the Blind and the State Training School for Boys, an institution for younger delinquents.

An important activity consisted in the direction of a survey of the hearing acuity of 5,575 rural school children of Hennepin County. This was carried on as a University of Minnesota project in cooperation with a county nursing project, both financed by CWA funds.

To devise a practical method for determining the performance characteristics of electric hearing devices, your Committee, in collaboration with the School of Electrical Engineering of the University of Minnesota, has begun a needed research project. This work promises to make possible information of special value to manufacturers, otologists and the hard of hearing public.

The activities of this Committee are disclosing a larger field of possible service to the medical profession, especially to the Fellows of this Academy.

During the past year our membership has been augmented by the addition of Dr. Burt R. Shurly and Dr. George M. Coates.

Respectfully submitted,

JOHN F. BARNHILL
GORDON BERRY
THOMAS E. CARMODY
E. H. CARY
GEORGE M. COATES
HOWARD V. DUTROW
AUSTIN A. HAYDEN
FREDERICK T. HILL
GEORGE L. TOBEY
BURT R. SHURLY
CLAUDE T. UREN
EDWARD F. ZIEGELMAN
HORACE NEWHART, *Chairman*

PRESIDENT WHEELER: Dr. Fenton will report for the Committee on Public Relations.

PUBLIC RELATIONS

Dr. Ralph A. Fenton: By direction of the Council at the Boston meeting, and acting under specific instructions from the President of the Program Committee meeting in January, your Committee on Public Relations secured abstracts and prepared 28 press releases and 3 radio broadcasts before reaching Chicago. The services of a press bureau approved by the American Medical Association were secured for the distribution of these releases, and 17 other press abstracts were prepared here, from papers received late, and discussing events of the session. Numerous special interviews were supervised by the Committee members. National services reporting on scientific medicine sent special representatives to the meeting.

It is felt that better control of lay interpretation of events on the Academy's program has been secured here than at any previous session.

In acordance with the rules of the American Medical Association, names of local physicians were excluded from all news stories, and the Committee desires to express its sincere appreciation for the cooperation of the editors and special writers for national and local press associations, local newspapers, and periodicals reporting on scientific medicine. Appreciation is also expressed to Dr. W. W. Bauer, Director of the Bureau of Health and Public Instruction of the Amercan Medical Association, for valuable hints on organization and for his arranging three public health broadcasts over local and national radio systems.

Public interest in the high standards of Academy membership, based upon certification of new members by the Boards, was evidenced by many telephoned and written inquiries and requests directed to the executive offices.

Your Committee has recommended to the Council the continuance of policies in effect at this meeting.

Respectfully submitted,

John J. Shea
Clarence King
Ralph A. Fenton, *Chairman*

President Wheeler: Dr. Gradle will report for the Committee on Ophthalmic Pathology.

OPHTHALMIC PATHOLOGY

DR. HARRY S. GRADLE: Mr. Chairman and Members: I should like to tell you what you are getting for your $500. In the first place, the Academy is no longer bearing the entire brunt of the work on ophthalmic pathology. By changing the name very slightly, we have been able to enlist the aid of the National Research Council, so now we are known as the Division of Ophthalmic Pathology of the American Registry of Pathology at the American Museum.

The following annual report of the Division of Ophthalmic Pathology of the American Registry of Pathology at the Army Medical Museum is submitted to the Committee on Pathology for the American Academy of Ophthalmology and Otolaryngology.

The numerical status of the Registry on July 1, 1934, was as follows:

TABLE I

Numerical Status of the Registry of Ophthalmic Pathology at the Army Medical Museum on July 1, 1934

	Eye Specimens Received and Reported	Eye Specimens Diagnosed When Received	Other Items Illustrations, Models, etc.	TOTAL
For the 8 yrs. ending December 31, 1928.....	977	629	1124	2730
For the year ending December 31, 1929.....	305	21	0	326
For the year ending December 31, 1930.....	275	202	5	482
For the year ending December 31, 1931.....	372	0	0	372
For the year ending December 31, 1932.....	309	0	2	311
For the year ending December 31, 1933.....	317	0	0	317
January 1, 1934 to July 1, 1934.....	161	2	0	163
TOTAL.....	2716	854	1131	4701

The number of eye specimens received for diagnosis has been very nearly the same each year for the past three years in spite of the generally bad economic situation throughout the United

States which materially reduced the number of accessions to the other registries during the same period.

Table Number II, appended to this report, shows the status of 237 primary melanotic tumors of the eye on January 1, 1934. Fifteen additional cases were received up to July 1, 1934, but are not included in the statistical table. This makes a total of 252 melanotic tumors on file in the Registry. Three cases of spindle B type died of metastases since the last report. The percentage of known deaths from each of the histologic types of melanoma of the eye on January 1, 1934, after the compilation of the annual follow-up reports is shown in Table III below.

TABLE III

Number and Per Cent of Deaths from Each Histologic Type of Melanotic Tumors of the Eye

Histologic Type	Total Cases	Number Lost	Number Followed	Died Tumors	Per Cent Dead
Spindle A	31	6	25	0	00.00
Spindle B	54	10	44	3	6.81
Epithelioid	17	3	14	3	21.42
Fascicular	12	0	12	3	25.00
Mixed	123	22	101	37	36.64
TOTAL.....	237	41	196	46	23.47

Up to the present time there have been no deaths from the purely spindle A type tumors and only a very small percentage of the spindle B type. Of the three deaths from spindle B type tumors reported last year, one died within three years of enucleation and the other two between three and five years after operation.

Research on the staining reaction of the melanotic tumors is being continued with a view of determining their origin and, if possible, of providing an additional standard for judging the prognosis in individual cases.

An analysis of all of the cases of retinoblastoma in the Registry was made in the last annual report. Tables IV and V, appended hereto, show the status of all cases received up to January 1, 1934. Seven additional specimens were received up to July 1, 1934, making a total of 102 cases of retinoblastoma in the Registry. The records pertaining to seventy-one of these are

adequate for inclusion in the statistical studies now being carried out. It is felt, however, that the number of cases in this group is still too small to form the basis of an article for publication.

A tabulation of the status of 60 cases of epithelial tumors of the ocular adnexa is made in Table Number VI, appended to this report.

During the past year twelve sets of twelve slides each were made for addition or replacements in the loan collections of histopathology of the eye. These 144 slides represent a total value of $324. The sets will be revised to include the more illustrative material after they have been returned to the Museum, following the course of ocular pathology to be given in Chicago during the month of September, 1934.

The $500 appropriation made by the Academy was used in the preparation of the slides for the loan collection and for the research in staining now being carried on at the Museum.

On the whole, the Registry of Ophthalmic Pathology is in excellent condition and the progress made fully justifies the expense of effort and money for its continuance.

V. H. Cornell
Major, Medical Corps, U.S.A.
Curator

TABLE II

Classification by Histologic Type of 237 Primary Melanotic Tumors of the Eye Showing Age, Distribution, Location, Degree of Pigmentation and Outcome after Enucleation on December 31, 1933

TYPE	AGE AT OPERATION						LOCATION			PIGMENT			STATUS AFTER OPERATION						DEATHS			
	Total Cases	20 to 39	40 to 49	50 to 59	60 to 69	70 and Over	Not Known	Uvea	Limbus	Orbital Extension from Uvea	Slight	Moderate	Abundant	3 years or Less		3 to 5 Years		5 Years and Over		Patient Lost	Tumor	Other	Unknown
														L	D	L	D	L	D				
Spindle A	31	2	9	9	6	4	1	31	0	4	12	10	9	13	3	3	0	5	1	6	0	3	1
Spindle B	54	11	8	14	20	1	0	53	1	10	28	23	3	30	1	4	2	3	2	10	3	4	0
Epithelioid	17	5	8	2	1	1	0	9	8	2	2	9	6	9	4	0	0	1	0	3	3	1	0
Fascicular	12	3	3	3	2	1	0	12	0	2	8	1	3	6	5	0	0	1	0	0	3	2	0
Mixed	123	17	20	37	30	15	5	121	2	37	16	47	60	31	34	13	8	13	4	22	37	5	2
TOTAL	237	38	48	65	59	22	6	226	11	55	66	90	81	89	47	20	10	23	7	41	46	15	3

L = Living D = Dead

TABLE IV

Status of 95 Cases of Retinoblastoma in the Registry of Ophthalmic Pathology, Army Medical Museum on December 31, 1933

Age at Operation	Male	Female	Sex unknown	Unilateral	Bilateral	Post-operative Duration Living — years: 1	1-2	2-3	3-4	4-5	Over 5	Total living	Dead	Status unknown	TOTAL
—1	3	5	0	3	5	1	1	1	2	0	1	6	1	1	8
1-2	8	7	0	9	6	3	4	1	0	0	2	10	4	1	15
2-3	16	7	0	17	6	1	0	0	0	0	5	6	14	3	23
3-4	15	7	1	17	6	4	0	1	0	0	3	8	12	3	23
4-5	5	4	1	9	1	0	0	1	0	0	2	3	5	2	10
5-6	1	0	0	1	0	0	0	0	0	0	0	0	1	0	1
6-11	4	3	0	5	2	1	1	0	0	0	0	1	2	3	7
Age Unknown	2	0	6	8	0	0	0	0	0	0	0	0	0	8	8
TOTAL	54	33	8	69	26	10	6	4	2	0	13	35	39	21	95

TABLE V

Tabulation by Mode of Extension of 71 Cases of Retinoblastoma of Which the Present Status Is Known December 31, 1933

Extension: Optic Nerve	Chor-oid	Ciliary Body	Sclera	Post-operative Duration Years: —1	2	3	4	5	5+	Total Living	Dead	TOTAL
—	—	—	—	2	2	2	0	0	9	15	3	18
+	+	+	+	0	1	0	0	0	0	1	15	16
+	+	—	—	1	1	1	1	0	0	4	5	9
+	+	—	+	1	0	0	0	0	1	2	4	6
+	+	+	—	0	0	0	0	0	0	0	6	6
+	—	—	—	1	1	1	1	0	1	5	4	9
—	+	—	—	0	1	0	0	0	2	3	0	3
+	—	+	+	0	0	0	0	0	0	0	1	1
+	—	—	+	1	0	0	0	0	0	1	1	2
—	—	+	—	1	0	0	0	0	0	1	0	1
TOTAL.....				7	6	4	2	0	13	32	39	71

TABLE VI

Tabulation of 60 Cases of Epithelial Tumors of the Ocular Adnexa
December 31, 1933

	AGE AT OPERATION							ORIGIN			LIVING YEARS POSTOPERATIVE						TOTAL LIVING	DEAD			NO REPORT SINCE OPERATION
	—20	20-39	40-49	50-59	60-69	70 & Over	Unknown	Lid	Conjunctiva	Cornea	—1	1-2	2-3	3-4	4-5	5+		Tumor	Others	Unknown	
Papilloma	1	2	0	0	2	1	1	2	5	0	1	0	1	1	0	1	4	0	0	0	3
Carcinoma	1	7	5	11	14	8	7	21	31	1	1	5	5	1	4	5	21	3	2	2	25
TOTAL	2	9	5	11	16	9	8	23	36	1	2	5	6	2	4	6	25	3	2	2	28

PRESIDENT WHEELER: If there are no objections, these eight reports will stand approved and will be placed on file.

Is there any unfinished business?

EXECUTIVE SECRETARY-TREASURER WHERRY: There is none on my desk, Mr. President.

New Business

PRESIDENT WHEELER: Is there any new business? If not, we will proceed to the election of new members, Dr. Wherry.

Election of Members

EXECUTIVE SECRETARY-TREASURER WHERRY: The Council has nominated the following candidates for membership:

Alexander, Lucian White, 1230 Maison Blanche Bldg., New Orleans, La., ALR

Allan, William Brewster, 100 E. Palisade Ave., Englewood, N. J., Op.

Avery, Elroy James, 11 N. Goodman St., Rochester, N. Y., ALR

Batson, Oscar V., 3502 Hamilton St., Philadelphia, Pa., ALR

Berke, Raynold N., 430 Union Street, Hackensack, N. J., 30 West 59th St., New York, N. Y., Op.

Blonder, Edwin J., 185 N. Wabash Ave., Chicago, Ill., ALR

Bostwick, Delazon Swift, 106 Linwood Ave., Ardmore, Pa., ALR

Bradley, Jeter Carroll, 815 Connecticut Ave., Washington, D. C., ALR

Bressler, Jacob Louis, 6254 S. Ashland Ave., Chicago, Ill., Op.

Brook, Jeffrey J., 231 W. Wisconsin Ave., Milwaukee, Wis., ALR

Brooks, Morris Jacob, 455 Ocean Ave., Brooklyn, N. Y., ALR

Brown, Lt. Comdr. Clarence J. (M.C.), U. S. N., Naval Dispensary, Navy Dept., Washington, D. C., ALR

Brumbaugh, John Daniel, 933 Second National Bldg., Akron, Ohio, ALR

Bucher, Walter M., 4187 Pearl Rd., Cleveland, Ohio, ALR

Clark, William Burton, 200 Carondelet St., New Orleans, La., Op.

Cress, William Joseph, 318 W. Market St., Pottsville, Pa., ALR

Danielson, Ralph Wesley, 258 Metropolitan Bldg., Denver, Colo., Op.

Davis, David, 1835 Eye St., N.W., Washington, D. C., ALR

Davis, Warren B., 135 S. Elizabeth St., Philadelphia, Pa., ALR

Dimmitt, Frank Whitenack, 195 Thayer St., Providence, R. I., ALR
Dodge, Warren M., Jr., Sanitarium, Battle Creek, Mich., Op.
Ellingwood, William Arthur, 420 Main St., Rockland, Me., ALR
Enlows, Ella Morgan Austin, 815 Connecticut Ave., Washington, D. C., ALR
Evans, William Hiram, 24 Wick Ave., Youngstown, Ohio, ALR
Ferris, Ralph Graves, 55 West Maple Ave., Birmingham, Mich., ALR
Fischer, Aubrey, 1835 Eye St., N.W., Washington, D. C., ALR
Fitzgerald, Fred J. C., 424 Madison Ave., New York, N. Y., Op.
Frey, Guernsey, 121 East 60th St., New York, N. Y., Op.
Garland, Frederick E., 483 Beacon St., Boston, Mass., LR
Gaudreau, Honore Edward, 293 Bridge St., Springfield, Mass., Op.
Goodall, Edwin Baker, 101 Bay State Road, Boston, Mass., Op.
Goodell, William, 121 Chestnut St., Springfield, Mass., ALR
Goodspeed, Harry Bancroft, 36 Pleasant St., Worcester, Mass., ALR
Graebner, Herbert, 45 Park Ave., New York, N. Y., ALR
Granberry, Carl, 1124 Maison Blanche Bldg., New Orleans, La., ALR
Griffin, Edwin Albert, 165 Hancock St., Brooklyn, N. Y., ALR
Guy, Loren Pritchard, 116 E. 62nd St., New York, N. Y., Op.
Hagaman, Adrian Van Dyke, 509 Lamar Life, Jackson, Miss., ALR
Hall, Sobisca Stalnaker, 95 Fairmont Ave., Fairmont, W. Va., ALR
Hallum, Alton Venson, 478 Peachtree St., Atlanta, Ga., Op.
Hickman, Warren R., 211 Third St., Logansport, Ind., ALR
Hildreth, Howard Rommel, 824 Metropolitan Bldg., St. Louis, Mo., Op.
Hirsch, Caesar, 667 Madison Ave., New York, N. Y., ALR
Hitz, John Barnard, 208 East Wisconsin Ave., Milwaukee, Wis., Op.
Hoover, Walter Boyd, 605 Commonwealth Ave., Boston, Mass., ALR
Houser, Karl Musser, 2010 Spruce St., Philadelphia, Pa., ALR
Hulka, Jaroslav H., 30-28 Thirty-Sixth St., Long Island City, N. Y., OALR
Hunt, William Taylor, Jr., 514 Penn St., Huntingdon, Pa., ALR
Ivy, Robert H., 1930 Chestnut St., Philadelphia, Pa., ALR

Jackson, Chevalier Lawrence, 3432 N. Broad St., Philadelphia, Pa., L

Keeler, Lansing Herman, 89-04 148th St., Jamaica, N. Y., Op.

Kelly, George F. J., Suite 700, N. E. Cor. 20th and Chestnut Sts., Philadelphia, Pa., Op.

Kershner, Warren Edglie, 117 Front St., Bath, Maine, ALR

Keyton, John Arthur, 108 E. Main St., Dothan, Ala., ALR

Kinney, Charles Edward, 10515 Carnegie Ave., Cleveland, Ohio, ALR

Kramer, Julius C., 130 E. Market St., Sandusky, Ohio, ALR

Leggett, Albert Earle, 614 Breslin Bldg., Louisville, Ky., Op.

Levy, Abram, 507 Church St., Bound Brook, N. J., Op.

Loewe, Walter Ralph, 1114 Madison Ave., New York, N. Y., Op.

Mann, Jacob John, 255 State Street, Perth Amboy, N. J., ALR

Maxwell, James Hoyt, 201 S. Main St., Ann Arbor, Mich., ALR

Meltzer, Philip Edward, 20 Charlesgate, West, Boston, Mass., ALR

Miller, Milton Valentine, 114 West Phil-Ellena St., Mt. Airy, Philadelphia, Pa., ALR

Miller, Saul, 8805 150th St., Jamaica, N. Y., Op.

Mills, Chester Rudolph, 520 Commonwealth Ave., Boston, Mass., ALR

Monson, LaFayette, 1801 Bush St., San Francisco, Calif., ALR

Moyer, John Sanford, 239 East 75th St., New York, N. Y., ALR

Murphy, William Edward, 390 Main St., Worcester, Mass., ALR

Neal, Phil Hawkins, 135 East 65th St., New York, N. Y., ALR

Newlands, William Alexander, 19 South Broadway, Tarrytown, N. Y., ALR

Newton, Morris H., Burrell Bldg., Little Falls, N. Y., Op.

O'Connor, Frederick James, 713 East Genesee St., Syracuse, N. Y., ALR

O'Connor, Philip Daniel, 30 North Michigan Ave., Chicago, Ill., Op.

Odom, Major Stanley Gibson (M. C., U. S. A.), Station Hospital, Fort Sheridan, Ill., OALR

Perdue, James D., Merchants National Bank Bldg., Mobile, Ala., Op.

Rigley, Edward Landon, Medical Arts Bldg., Omaha, Nebr., ALR

Riker, Aaron Dudley, 1012 Riker Bldg., Pontiac, Mich., Op.

Riwchun, Meyer Herbert, 468 Delaware Ave., Buffalo, N. Y., Op.

Sawyer, Leroy Lee, Jr., The Farragut Medical Bldg., Washington, D. C., ALR

Schillinger, Raphael, 897 Park Place, Brooklyn, N. Y., ALR
Schmidt, Herbert G., 238 W. Wisconsin Ave., Milwaukee, Wis., ALR
Seidel, Urban Daniel, 925 Second National Bldg., Akron, Ohio, ALR
Shapira, Theodore Morrison, 58 E. Washington, St., Chicago, Ill., Op.
Sherman, R. Newton, 400 Phoenix Bldg., Bay City, Mich., OALR
Simmons, Major Ralph Hayward (M. C.), 2nd Corps Area Hospital, Fort Jay, Governor's Island, N. Y., ALR
Slack, Walter Knapp, 308 Eddy Bldg., Saginaw, Mich., ALR
Stauffer, Fred Leaver, 707 Medical Arts Bldg., Salt Lake City, Utah, ALR
Stokes, Harry Bayliss, 26 S. Goodman St., Rochester, N. Y., ALR
Taylor, Joseph W., 706 Franklin St., Tampa, Florida, ALR
Thygeson, Phillips, Dept. of Oph., University Hosp., Iowa City, Iowa, Op.
Tippin, Ernest E., 601 First Nat'l Bldg., Wichita, Kansas, ALR
Waldeck, Edward Alvin, 238 W. Wisconsin Ave., Milwaukee, Wis., Op.
Wechsler, Abraham Hirsch, 340 New York Ave., Brooklyn, N. Y., ALR
Whelan, William Francis, 2015 Walnut St., Philadelphia, Pa., ALR
White, James Watson, 15 Park Ave., New York, N. Y., Op.
Wilkins, Samuel Henry, 270 Commonwealth Ave., Boston, Mass., Op.
Wilkinson, Richard Wallace, 1408 L St., N.W., Washington, D. C., ALR
Winkler, Herman, 224 Thayer St., Providence, R. I., ALR

Sir, I move that these ninety-eight candidates be elected to membership.

The motion was regularly seconded, put to a vote and carried unanimously.

Election of Officers

PRESIDENT WHEELER: The next order of business is the election of officers.

EXECUTIVE SECRETARY-TREASURER WHERRY: The Council desires to nominate the following officers for the year 1935:

President

DR. WELLS P. EAGLETON.....Newark

President-Elect

DR. FRANK E. BURCH.....St. Paul

First Vice-President

DR. W. THORNWALL DAVIS.....Washington

Second Vice-President

DR. SAMUEL IGLAUER.....Cincinnati

Third Vice-President

DR. M. F. WEYMANN.....Los Angeles

Comptroller

DR. SECORD H. LARGE.....Cleveland

Executive Secretary-Treasurer

DR. W. P. WHERRY.....Omaha

Associate Secretaries

Ophthalmology

DR. WILLIAM L. BENEDICT.....Rochester, Minn.

Otolaryngology

DR. JOHN L. MYERS.....Kansas City, Mo.

Instruction (Ophthalmology)

DR. HARRY S. GRADLE.....Chicago

Instruction (Otolaryngology)

DR. WILLIAM V. MULLIN.....Cleveland

Miscellaneous Activities

DR. RALPH A. FENTON.....Portland, Ore.

Member of Ophthalmic Board (to finish unexpired term of Dr. Phinizy Calhoun, Atlanta, resigned)

DR. THOMAS D. ALLEN.....Chicago

Member of Ophthalmic Board (for four years)

DR. EVERETT GOAR.....Houston, Texas

Member of Council

DR. ROBERT FORGRAVE.....St. Joseph, Mo.

DR. H. P. MOSHER: Mr. President and Members of the Society: I have the honor to move the election of the members whose names have just been read, as officers of this society.

DR. SHEA: Mr. President, may I second that?

PRESIDENT WHEELER: Dr. Mosher has moved the election of the officers just nominated, and the motion has been seconded by Dr. Shea. Those in favor of the motion for the election of these officers say "aye"; opposed, "no." It is unanimously carried, and they are declared elected.

Dr. Gradle, will you be kind enough to escort Dr. Eagleton, our new President, to the platform?

The audience arose and applauded as President-Elect Eagleton was escorted to the platform.

PRESIDENT-ELECT EAGLETON: This is no time for a speech. I just simply want to say the honor I feel in being elected and to express at this time my thanks to the outgoing President for the uniform kindness and courtesy that he has shown me, and my profound admiration for the secretarial staff of this organization. I have never seen anything (and I have been with many organizations) that equals this in efficiency, good will and desire to be of service.

Selection of 1935 Convention City

PRESIDENT WHEELER: The next item of business is the selection of the 1935 convention city.

EXECUTIVE SECRETARY-TREASURER WHERRY: For those who do not know, in 1910, three small western societies were amalgamated at a meeting in Cincinnati into the American Academy of Ophthalmology and Otolaryngology.

Because two of the societies were, shall I say, financially embarrassed, and the Western Society had a little money in the treasury, the Western more or less absorbed the other two.

In 1935, twenty-five years will have elapsed since that association, and the Academy has not been back to Cincinnati in this interval.

The Council thought it a very fitting occasion to celebrate the twenty-fifth anniversary of the actual formation of the Academy in the city where it was created.

Therefore, I have been instructed by the Council to move, sir, that Cincinnati be selected as the convention city for 1935.

DR. F. R. SPENCER: I second the motion.

PRESIDENT WHEELER: It has been moved and seconded that Cincinnati be the city selected for the 1935 convention. Those in favor of this motion say "aye"; opposed "no." It is carried.

Is there any further business to come before the meeting? If not, the meeting will stand adjourned.

The meeting adjourned at six-thirty o'clock.

Directory

The American Academy
of Ophthalmology and Otolaryngology

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Thirty-Ninth Annual Meeting
Chicago . . . 1934

Alphabetical List of Members

* Attended 1934 meeting. Number in parentheses indicates year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

NOTICE: By action of the Council at the 1934 meeting:

- Fellows who have paid dues for thirty (30) years continuously will automatically become Life Members, with dues remitted.
- Fellows who have paid dues continuously for twenty-five (25) years will be termed Senior Members and their dues will be one-half the usual amount; at the present time, five dollars ($5.00) anually.
- All other Fellows (except Honorary) will be classified as Junior Members.

HONORARY FELLOWS

*Beck, Joseph C. ('01) . . . . . 185 N. Wabash Ave., Chicago, Ill., ALR

Birkett, H. S. ('06) . . . . 1190 Mountain St., Montreal, Canada, ALR

Dundas-Grant, Sir James. . . . . 148 Harley St., W. 1, London, Eng.

Elliot, Lt.-Col. R. H. ('13)
54 Welbeck St., Cavendish Sq., W., London, Eng.

Elschnig, Prof. A. ('12) . Ferdinandstrasse 10, Prague, Czechoslovakia

Graves, Basil ('26)
11 Devonshire Place, Upper Wimpole St., W. 1, London, Eng.

Gray, Albert A. ('11) . . . . . Glasgow, Scotland

de Grósz, Prof. Emil ('30) . . . . . Budapest, VIII, Maria, V. 39, Op.

*Jackson, Edward ('02) . . . . . 314 Fourteenth St., Denver, Colo., Op.

Johnson, Lindsay ('05) . Britannia Bldg., West St., Durban, Natal, Africa

Kirkpatrick, Lt.-Col. Henry ('24)
54 Welbeck St., W. 1, London, Eng.

Koller, Carl ('34) . . . . . 30 E. 58th St., New York, N. Y., Op.

*Lauber, Prof. Dr. Hans ('34) . . . Aleja Roz 8, Warsaw, Poland, Op.

Lawford, J. B. ('08) . . . Sagamore, New Road, Ashtead, Surrey, Eng.

Lister, Sir William T. ('23) . 24 Devonshire Pl., W. 1, London, Eng.

Nagar, Prof. F. ('25) . . . . . Freiestrasse 20, Zurich, Switzerland

Patterson, Maj. Gen. Robert U. ('33)
U. S. Army, Office of Surgeon General, Washington, D. C., ALR

Tilley, Herbert ('29) . . University College Hosp., London, Eng., Op.

Van der Hoeve, Prof. J. ('21) . Rijnsburgerweg, 6 A, Leiden, Holland

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Old Merchants Tower, Battle Creek, Mich., Op.
Smith, Dorland('10) . . 836 Myrtle Ave., Bridgeport, Conn., Op.A
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HUGHES, W. F. .....401 Hume-Mansur Bldg., Op.
KEARBY, D. O. .....Hume-Mansur Bldg., ALR
LARKIN, BERNARD J. .....Hume-Mansur Bldg., Op.
LEASURE, JOHN K. .....Hume-Mansur Bldg., ALR
LINGEMAN, EDWARD LAUREL .....406 Pennway Bldg., ALR
LITTELL, J. J. .....Hume-Mansur Bldg., ALR
MCCASKEY, C. H. .....Continental Bank Bldg., ALR
MASTERS, ROBERT J. .....23 E. Ohio St., Op.
MOLT, WM. F. .....529 Bankers Trust Bldg., ALR
MUELLER, LILLIAN BARBARA .....Continental Bank Bldg., ALR
NEWCOMB, JOHN R. .....410 Hume-Mansur Bldg., Op.
OVERMAN, F. V. .....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A. .....445 Bankers Trust Bldg., OALR
ROW, GEORGE S. .....906 Hume-Mansur Bldg., Op.
TOMLIN, W. S. .....Hume-Mansur Bldg., ALR
VAN OSDOL, HARRY A. .....Chamber of Commerce Bldg., ALR
WHITAKER, J. .....Hume-Mansur Bldg., OALR
WRIGHT, J. WM. .....Hume-Mansur Bldg., ALR

KOKOMO

ADAMS, CHAS. JOSEPH .....Armstrong-Landon Bldg., OALR

LOGANSPORT

EGAN, BURTON WRIGHT .....408 North St., OALR
HICKMAN, WARREN R. .....211 Third St., ALR

MARION

BRAUNLIN, WM. H. .....Marion Nat'l Bank Bldg., OALR
MILLER, C. E. .....Western Reserve Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M. .....701 Washington St., OALR

RICHMOND

HOLLAND, EMORY ED. ..... 501 S. Main St., Op.

SOUTH BEND

BOONE, J. C. ..... Sherland Bldg., ALR
BOYD-SNEE, HARRY ..... 716 J. M. S. Bldg., ALR
CASSADY, J. VERNAL ..... 654 Associates Bldg., OALR
EDWARDS, SLOCOMB R. ..... Christman Bldg., OALR
LENT, E. J. ..... 122 N. Lafayette Blvd., OALR
SAVERY, C. E. ..... 206 Sherland Bldg., OALR

TERRE HAUTE

ALLEN, ORRIS T. ..... 422 Rose Dispensary Bldg., OALR
GILLUM, J. R. ..... 221 S. 6th St., OALR
MILLER, WM. H. ..... Tribune Bldg., ALR
RHEIN, ALFRED E. ..... Tribune Bldg., ALR
STEWART, WALTER E. ..... Tribune Bldg., OALR

UNION CITY

RUBY, F. M. ..... Physicians' Bldg., OALR

WARSAW

HOWARD, C. NORMAN ..... Warsaw Clinic, OALR

IOWA

AMES

DYER, BENJAMIN G. ..... ALR

BURLINGTON

LA FORCE, E. F. ..... Iowa State Bank Bldg., OALR
YOUNG, H. B. ..... 204 Jefferson St., OALR

CEDAR RAPIDS

BAILEY, FRED W. ..... Merchants Nat'l Bank Bldg., OALR
IVINS, H. M. ..... 815 Merchants Nat'l Bank Bldg., OALR

CLINTON

WEIH, E. P. ..... 217 Fifth Ave. South, OALR

COUNCIL BLUFFS

DEAN, F. W. ..... 532 First Ave., OALR
HOWARD, LLOYD G. ..... Council Bluffs Clinic, OALR
MAIDEN, S. D. ..... 403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A. ..... Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT ..... 201 West Second St., ALR
HARKNESS, GORDON F. ..... Putnam Bldg., OALR
HOFMANN, W. P. ..... 707 Kahl Bldg., OALR
LAMB, HARRY HOWE ..... Davenport Bank Bldg., ALR

DES MOINES

JONES, CECIL C. ..... Equitable Bldg., OALR
LYNCH, ROBERT J. ..... Equitable Bldg., ALR
MUMMA, CLAUDE S. ..... Hotel Fort Des Moines, ALR
PEARSON, WM. W. ..... 417 Bankers Trust Bldg., OALR

DUBUQUE

THOMPSON, HOWARD E. ..... 1100 Main St., OALR

FORT DODGE

CHASE, S. B. ..... Carver Bldg., OALR

IOWA CITY

ALBRIGHT, GEORGE C. ..... 102 S. Clinton S., ALR
BOILER, W. F. ..... Dey Bldg., OALR
LIERLE, DEAN MCA. ..... University Hospital, ALR
MOORE, PAUL MCNARY, JR. ..... University Hospital, ALR
O'BRIEN, C. S. ..... University Hospital, Op.
RUTHERFORD, C. W. ..... Univ. Hosp., Dept. Ophth., Op.
THYGESON, PHILLIPS ..... Dept. of Ophth., Univ. Hosp., Op.

KEOKUK

LAPSLEY, ROBERT M. ..... Masonic Temple, OALR

LE MARS

JOYNT, M. J. ..... Royal Bldg., ALR

MARSHALLTOWN

COBB, EDWIN ..... Masonic Temple Bldg., OALR
FRENCH, R. F. ..... Masonic Temple Bldg., OALR
WOLFE, OTIS R. ..... 415 Masonic Temple, Op.

MASON CITY

FALLOWS, HOWARD D. ..... I.O.F. Bldg., OALR
O'BRIEN, STEVE A. ..... First Nat'l Bank Bldg., OALR

MUSCATINE

GOAD, R. R. ..... Laurel Bldg., Op.

NEWTON

ENGLE, H. P. ..... 212 West First Ave., OALR

OTTUMWA

TAYLOR, CHARLES B. ..... 120 E. Second St., ALR

SHENANDOAH

MALOY, WAYLAND H. ..... 608½ W. Sheridan Ave., Op.

SIOUX CITY

DECKER, J. C. ..... Frances Bldg., OALR
GITTINS, T. R. ..... 401 Davidson Bldg., OALR
NAFTZGER, J. B. ..... Davidson Bldg., OALR
REEDER, J. E. ..... Davidson Bldg., OALR
SALLANDER, F. W. ..... New Orpheum Bldg., OALR
SECOY, F. L. ..... 301 Davidson Bldg., OALR
TRIPP, LEROY R. ..... Security Bldg., OALR

WATERLOO

BENDER, H. A. ..... Black Bldg., ALR
JOYNT, A. J. ..... Black Bldg., OALR
REULING, F. H. ..... Black Bldg., OALR
RUSSELL, R. E. ..... 722 Caward Bldg., OALR

WAVERLY

ROBINSON, R. E. ..... 205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J. ..... Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B. ..... Terminal Bldg., Op.

CONCORDIA

ROBERTSON, EDW. N. ..... New Caldwell Bldg., OALR

ELDORADO

SHULTS, E. J. G. ..... Haines Bldg., OALR

EMPORIA

GRANGER, W. B. ..... Gazette Bldg., OALR
TRIMBLE, CLARENCE S. ..... Gazette Bldg., OALR

HUTCHINSON

GREEVER, W. B. ..... 100 N. Main St., OALR
SCALES, H. L. ..... Rorabaugh Wiley Bldg., OALR
SCHRANT, J. H. ..... 14 West First St., OALR

KANSAS CITY

LIDIKAY, C. J. ..... 903 N. Seventh St., OALR
SPAKE, LAVERNE B. ..... Brotherhood Bldg., ALR

LAWRENCE

POWELL, LYLE S. ..... 833½ Mass. St., OALR

TOPEKA

ALKIRE, H. L. ..... 614 Kansas Ave., OALR
ALLEN, GEORGE V. ..... 835 Kansas Ave., OALR

ALLEN, G. HENRY ..... Mills Bldg., OALR
DILLON, IRA H. ..... A. T. & S. F. Ry. Hospital, Op.
REED, WM. W. ..... Mills Bldg., ALR

WICHITA

BROWNELL, MORTON EMMONS ..... 105 N. Main St., Op.
CARTER, E. D. ..... Central Bldg., ALR
CHENEY, JAMES W. ..... 702 Schweiter Bldg., OALR
GILLETT, WILBUR G. ..... 201½ N. Main St., Op.
GSELL, J. F. ..... 911 Beacon Bldg., OALR
MAGGARD, D. I. ..... 715 Orpheum Bldg., OALR
SEYDELL, E. M. ..... 1030 First Nat'l Bank Bldg., ALR
TIPPIN, ERNEST E. ..... 601 First Nat'l Bank Bldg., ALR
WEAVER, T. W. ..... 201½ N. Main St., Op.

WINFIELD

RALLS, C. T. ..... First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY ..... OALR

COVINGTON

BLEDSOE, R. W. ..... 1005 Madison Ave., OALR

GLASGOW

WELDON, WILLIAM A. ..... Op.

HARLAN

GUNN, MILUS LYDELL ..... Smith Bldg., ALR

LEXINGTON

ARMSTRONG, R. M. ..... Fayette Bank Bldg., OALR
DEWEESE, CLARENCE ..... 201 West Second St., OALR
MARKS, SAMUEL BLACKBURN ..... Security Trust Bldg., ALR
OFFUTT, W. N. ..... 406 Security Trust Bldg., OALR
YATES, EARL C. ..... 190 N. Upper St., ALR

LOUISVILLE

BASS, A. L. ..... Heyburn Bldg., OALR
DABNEY, S. G. ..... Starks Bldg., OALR
DEAN, WALTER ..... 300 Francis Bldg., OALR
DULANEY, OCTAVIUS ..... 811 Brown Bldg., ALR
HALL, G. C. ..... 705 Brown Bldg., OALR
HEITGER, J. D. ..... Heyburn Bldg., OALR
LEGGETT, ALBERT E. ..... 614 Breslin Bldg., Op.
PFINGST, A. O. ..... Heyburn Bldg., Op.
SHAFER, J. J. ..... Starks Bldg., OALR
TOWNES, C. DWIGHT ..... Heyburn Bldg., Op.
VAUGHAN, G. E. ..... 711 S. Third St., OALR
WOLFE, CLAUDE T. ..... Heyburn Bldg., OALR

PADUCAH

PACE, ERRETT ..... City National Bank Bldg., ALR
REYNOLDS, H. G. ..... City National Bank Bldg., OALR

RICHMOND

DUNN, MURISON ..... OALR

LOUISIANA

LAKE CHARLES

MOSS, OLIN W. ..... Weber Bldg., ALR

NEW ORLEANS

ALEXANDER, LUCIAN W. ..... 1230 Maison Blanche Bldg., ALR
BAHN, CHAS. A. ..... Pere Marquette Bldg., Op.
BLUM, HENRY N. ..... American Bank Bldg., Op.
BREWSTER, H. F. ..... Union Indemnity Bldg., ALR
BROWN, M. EARLE ..... 921 Canal St., Op.
CLARK, WM. B. ..... 200 Carondelet St., Op.
COLLIER, GEO. BENJAMIN ..... 1317 Whitney Bldg., ALR
FUCHS, VALENTINE HENRY ..... Chaille Bldg., ALR
GRANBERRY, CARL ..... Maison Blanche Bldg., ALR
HASPEL, M. DAVID ..... Maison Blanche Bldg., OALR
HUME, JOHN R. ..... Maison Blanche Bldg., ALR
LANDFRIED, C. J. ..... 607 Baronne Bldg., ALR
LEJEUNE, F. E. ..... 632 Maison Blanche Bldg., ALR
SMITH, VICTOR C. ..... American Bank Bldg., Op.
TAQUINO, GEORGE J. ..... 208 Audubon Bldg., ALR
WAGNER, W. A. ..... 200 Carondelet St., ALR
WEIL, A. I. ..... 3503 Prytania St., ALR
WOMACK, DAVID R. ..... Maison Blanche Bldg., ALR

SHREVEPORT

LARUE, C. L. ..... Highland Clinic, OALR
MOISE, ALLYN B. ..... Medical Arts Bldg., ALR
SCALES, J. L. ..... Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W. ..... Masonic Temple, OALR

BATH

KERSHNER, W. E. ..... 117 Front St., ALR

LEWISTON

CHAFFERS, WM. H. ..... 190 Bates St., ALR

PORTLAND

BEACH, S. JUDD ..... 704 Congress St., Op.
CUMMINGS, GEORGE O. ..... 47 Deering St., ALR

FISHER, STANWOOD E. ..... 192 State St., ALR
GILBERT, FRANK Y. ..... 148 Park St., OALR
GORDON, CHAS. H. ..... 46 Deering St., ALR
HASKELL, ALFRED W. ..... 142 High St., Op.
HOLT, E. E., JR. ..... 723 Congress St., Op.
MCADAMS, WM. R. ..... 704 Congress St., Op.
O'SULLIVAN, T. J. ..... 10 Pine St., ALR
SPALDING, J. A. ..... 627 Congress St., Op. A

ROCKLAND

ELLINGWOOD, WM. A. ..... 420 Main St., ALR

WATERVILLE

HILL, FREDERICK T. ..... Professional Bldg., ALR
HILL, HOWARD F. ..... Professional Bldg., Op.

MARYLAND

ANNAPOLIS

WELCH, ROBERT S. G. ..... 86 State Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR. ..... 300 N. Charles St., OALR
FRIEDENWALD, HARRY ..... 1212 Eutaw Pl., Op. A
FRIEDENWALD, J. S. ..... 1212 Eutaw Pl., Op.
GOLDBACK, L. J. ..... 1012 N. Charles St., Op.
KEMLER, JOS. I. ..... 1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON ..... 104 W. Madison St., OALR
PETERMAN, H. E. ..... 512 Cathedral St., OALR
TARUN, WILLIAM ..... 104 W. Madison St., Op.

CUMBERLAND

JONES, E. L. ..... First National Bank Bldg., OALR
ROBINSON, H. T. ..... Medical Bldg., OALR
SHARRETT, GEORGE O. ..... 119 Bedford St., OALR

MASSACHUSETTS

BEVERLY

ODENEAL, THOMAS HELM ..... 21 Broadway, OALR

BOSTON

BRYANT, ALICE GERTRUDE ..... 502 Beacon St., ALR
CAHILL, HARRY P. ..... 520 Commonwealth Ave., ALR
CASTEN, VIRGIL G. ..... 520 Commonwealth Ave., Op.
CHENERY, W. E. ..... 377 Commonwealth Ave., ALR
CLINE, SAMUEL ..... 353 Commonwealth Ave., ALR
COWAN, CLYDE R. ..... Hotel Buckminster, ALR
DUNPHY, ED. B. ..... 101 Bay State Road, Op.
EVANS, MAURICE GEO. ..... 311 Commonwealth Ave., ALR
FREEDMAN, L. M. ..... 475 Commonwealth Ave., ALR

GARLAND, FREDERICK E. .....483 Beacon St., LR
GOODALL, EDWIN BAKER .....101 Bay State Road, Op.
GREENWOOD, ALLEN .....82 Commonwealth Ave., Op.
HALEY, WM. THOS. .....531 Beacon St., ALR
HARTSHORNE, ISAAC .....22 Commonwealth Ave., Op.
HOOVER, WALTER BOYD .....605 Commonwealth Ave., ALR
HURLEY, E. D. .....419 Boylston St., Op.
INGLIS, HARRY J. .....43 Bay State Road, ALR
JOHNS, JUANITA PEARL .....313 Commonwealth Ave., Op.
KAZANJIAN, V. H. .....475 Commonwealth Ave., LR
KELLEY, VINCENT J. .....520 Commonwealth Ave., ALR
KLEINERT, MARGARET NOYES .....82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON .....520 Commonwealth Ave., ALR
LANCASTER, W. B. .....520 Commonwealth Ave., Op.
LEWIS, J. P. .....543 Boylston St., ALR
LOWELL, W. HOLBROOK .....82 Commonwealth Ave., Op.
LURIE, MOSES H. .....483 Beacon St., ALR
MELTZER, PHILIP E. .....20 Charlesgate West, ALR
MILLS, CHESTER R. .....520 Commonwealth Ave., ALR
MOSHER, HARRIS P. .....828 Beacon St., ALR
MYSEL, PHILIP .....311 Commonwealth Ave., ALR
POIRIER, GEORGE H. .....60 Bay State Road, ALR
PORTER, CHAS. T. .....520 Commonwealth Ave., ALR
QUINCY, J. E. .....270 Commonwealth Ave., ALR
REGAN, JAS J. .....520 Commonwealth Ave., Op.
RICE, G. B. .....99 Bay State Road, ALR
RICHARDS, L. G. .....319 Longwood Ave., ALR
ROWLAND, W. D. .....220 Clarendon St., Op.
SCHALL, LEROY A. .....270 Commonwealth Ave., ALR
SKIRBALL, J. J. .....353 Commonwealth Ave., Op.
SMYTH, DUNCAN C. .....375 Commonwealth Ave., ALR
TERRY, THEODORE L. .....128 Newbury St., Op.
THOMPSON, PETER H. .....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE .....9 Massachusetts Ave., Op.
TOBEY, GEORGE L., JR. .....270 Commonwealth Ave., ALR
WELLS, DAVID W. .....Hotel Westminster, Op.
WILKINS, SAMUEL HENRY .....270 Commonwealth Ave., Op.

CLINTON

MONAHAN, J. A. .....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R. .....151 Rock St., ALR

FRAMINGHAM

TOMB, EVERETT H. .....129 Concord St., ALR

GARDNER

BONE, HERMAN D. .....19 Pleasant St., OALR

GLOUCESTER

DEXTER, MILTON A. ..... 48 Pleasant St., ALR
ODENEAL, THOMAS H. ..... 40 Middle St., OALR

GREENFIELD

MILLETT, FRANK A. ..... 58 Federal St., OALR

HOLYOKE

BURKE, JOHN R. ..... 207 Elm St., OALR
HUSSEY, E. J. ..... 245 Maple St., OALR

LAWRENCE

CONLON, F. A. ..... 301 Essex St., OALR

LOWELL

WIGGIN, WM. IRVING ..... 202 Central St., ALR

LYNN

JONES, E. W. ..... 44 Atlanta St., Op. A
O'REILLY, W. F. ..... 44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F. ..... 232 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D. ..... 187 Main St., ALR
DOW, FRANK E. ..... 4 West St., Op.

PALMER

MOORE, G. A. ..... 353 Main St., OALR

PITTSFIELD

THOMPSON, G. H. ..... 74 North St., OALR

SALEM

HENNESSEY, W. W. ..... 333 Essex St., OALR

SPRINGFIELD

BOSS, EUGENE G. ..... 1537 Main St., Op.
BYRNES, H. F. ..... 67 Chestnut St., OALR
CARLETON, RALPH ..... 10 Temple St., Op.
GALLAGHER, JOHN H. C. ..... 1537 Main St., ALR
GAUDREAU, HONORE E. ..... 293 Bridge St., Op.
GOODELL, WILLIAM ..... 121 Chestnut St., ALR
IRWIN, VINCENT J., JR. ..... 1634 Main St., OALR
IRWIN, V. J., SR. ..... 1634 Main St. ,OALR
SEGAL, SAMUEL, JR. ..... 1562 Main St., Op.

TAUNTON

BAKER, HARRY B. ..... 59 Main St., OALR

WALTHAM

LEWIS, J. P. ..... 681 Main St., ALR

WORCESTER

BERRY, GORDON ..... 36 Pleasant St., ALR
CROISSANT, CHAS. A. ..... 390 Main St., ALR
CROSS, A. E. ..... 390 Main St., Op.
ESTABROOK, CHAS. T. ..... 857 Pleasant St., OALR
GIBBY, HAROLD JAMES ..... 27 Elm St., ALR
GOODSPEED, HARRY B. ..... 36 Pleasant St., ALR
HOLZER, WM. F. ..... 36 Pleasant St., Op.
MESSIER, A. EUGENE ..... 390 Main St., OALR
MURPHY, WILLIAM E. ..... 390 Main St., ALR
RICE, JOHN E. ..... 390 Main St., OALR

MICHIGAN

ALMA

HOWELL, D. M. ..... Polasky Bldg., ALR

ALPENA

NEWTON, WM. B. ..... 201 N. Second Ave., OALR

ANN ARBOR

BARR, ALBERT S. ..... First Nat'l Bank Bldg., Op.
FRALICK, F. BRUCE ..... First Nat'l Bank Bldg., Op.
FURSTENBERG, A. C. ..... First Nat'l Bank Bldg., ALR
MAXWELL, JAMES H. ..... 201 S. Main St., ALR

BATTLE CREEK

DODGE, WARREN M., JR. ..... Sanitarium, Op.
FRASER, R. H. ..... 25 W. Michigan Ave., ALR
HAUGHEY, WILFRED H. ..... Post Bldg., OALR
SLEIGHT, R. D. ..... Old Merchants Tower Bldg., Op.
WENCKE, CARL G. ..... Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H. ..... Bay City Bank Bldg., OALR
SHERMAN, R. NEWTON ..... 400 Phoenix Bldg., OALR

BENTON HARBOR

WESTERVELT, H. O. ..... 239 Pipestone St., ALR

BIRMINGHAM

FERRIS, RALPH G. ..... 55 W. Maple Ave., ALR

DETROIT

AMBERG, EMIL ..... 622 Maccabee Bldg., ALR
BARNETT, SAUL E. ..... 25 Parsons St., ALR
BEATTIE, ROBERT ..... David Whitney Bldg., OALR
BEGLE, HOWELL L. ..... David Whitney Bldg., Op.
BENTLEY, NEIL I. ..... David Whitney Bldg., OALR

BERNSTEIN, E. J. ..... 10 Peterboro, OALR
BROWN, AUDREY O. ..... 5057 Woodward, OALR
CAMPBELL, DON M. ..... David Whitney Bldg., OALR
CAMPBELL, DUNCAN A. ..... David Whitney Bldg., OALR
CAMPBELL, MALCOLM D. ..... 269 Rowena St., OALR
CARTER, JOHN M. ..... David Whitney Bldg., ALR
CLIFFORD, T. P. ..... David Whitney Bldg., OALR
COHOE, D. A. ..... 13535 Woodward Ave., ALR
DEFNET, WM. A. ..... 28 Adams Ave. West, ALR
DOWLING, HARVEY E. ..... Eaton Tower, Op.
FOWLER, WM. ..... Maccabee Bldg., OALR
GLOWACKI, BEN FRANCIS ..... Maccabee Bldg., OALR
GOUX, RAYMOND SALOT ..... 545 David Whitney Bldg., OALR
GRANT, LEE E. ..... David Whitney Bldg., OALR
HALE, ARTHUR STANLEY ..... 1609 Eaton Tower, OALR
HARRELL, VOSS ..... Hofman Bldg., ALR
HEATH, PARKER ..... David Whitney Bldg., Op.
HOWELL, DONALD MACLEAN ..... David Whitney Bldg., ALR
HUGHES, RAY W. ..... 1501 David Whitney Bldg., OALR
KEATING, T. F. ..... 1042 Maccabee Bldg., OALR
KREUTZ, GEORGE C. ..... Henry Ford Hospital, ALR
MCCLELLAND, CARL C. ..... 1857 David Whitney Bldg., OALR
MAIRE, LEWIS E. ..... 710 Kresge Bldg., OALR
MERCER, R. E. ..... David Whitney Bldg., LR
MINER, S. G. ..... 2641 E. Jefferson Ave., ALR
MORTON, J. B. ..... 90 E. Warren Ave., OALR
MUNSON, F. T. ..... 2201 E. Jefferson Ave., ALR
O'HORA, B. A. ..... 8-266 General Motors Bldg., ALR
PARKER, WALTER R. ..... David Whitney Bldg., Op.
PEIRCE, HOWARD W. ..... David Whitney Bldg., ALR
POOS, E. E. ..... Fisher Bldg., OALR
POTTER, WILLIS A. ..... 7450 Grand River Ave., ALR
RENAUD, G. L. ..... 510 Joseph Campan Ave., OALR
RYERSON, F. L. ..... Fine Arts Bldg., OALR
SANDERSON, H. H. ..... David Whitney Bldg., OALR
SCARNEY, HERMAN D. ..... 2501 W. Grand Blvd., Op.
SHURLY, BURT R. ..... 62 Adams Ave. W., ALR
SIMPSON, H. LEE ..... David Whitney Bldg., ALR
SUMMERS, WM. S. ..... 1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION ..... General Motors Bldg., ALR
WALKER, CHAS. C. ..... 604 Maccabee Bldg., Op.
WENDEL, J. S. ..... 744 David Whitney Bldg., ALR
WHITNEY, E. L. ..... Henry Ford Hospital, ALR
WILLSON, W. W. ..... 2204 Eaton Tower, OALR
WOODWORTH, WILLIAM P. ..... 2994 E. Grand Blvd., OALR
WILKINSON, A. P. ..... 664 Fisher Bldg., ALR

FLINT

HUBBARD, WM. B. ..... Dryden Bldg., ALR
STEVENSON, WM. W. ..... Kresge Bldg., ALR

GRAND RAPIDS

DEAN, ALFRED ..... 308 Medical Arts Bldg., Op.
DEWAR, MURRAY M. ..... Medical Arts Bldg., Op.
GRANT, P. T. ..... Medical Arts Bldg., OALR
HEETDERKS, D. R. ..... Medical Arts Bldg., ALR
LAIRD, ROBT. G. ..... Metz Bldg., Op.
MCRAE, J. H. ..... Medical Arts Bldg., Op.
MOLL, ARTHUR M. ..... Powers Bldg., ALR
ROBERTSON, F. D. ..... Ashton Bldg., OALR
ROLLER, LOUIS A. ..... 446 Paris Ave., S.E., Retired
SMITH, FERRIS ..... Grand Rapids Clinic, ALR
SNAPP, CARL F. ..... Medical Arts Bldg., ALR

GROSSE POINTE VILLAGE

MERKEL, CHAS. C. ..... 279 Rivard Blvd., ALR

HOLLAND

HUIZINGA, J. G. ..... Peoples State Bank Bldg., OALR

IONIA

SEDGWICK, O. W. ..... OALR

IRON MOUNTAIN

BOYCE, G. H. ..... Box 417, OALR

JACKSON

HARDIE, GEORGE C. ..... 290 W. Michigan Ave., ALR
MCGARVEY, WM. EDWARD. ..... Jackson City Bank Bldg., ALR
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ANDREWS, H. D. .....33 Allen St., Op.
BENNETT, ARTHUR G. .....150 N. Pearl St., Op.
BEYER, LOUIS J. .....449 Delaware Ave., ALR
BLAAUW, E. E. .....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE .....468 Delaware Ave., ALR
BROWN, C. M. .....510 Delaware Ave., ALR
COTT, C. C. .....483 Delaware Ave., ALR
COWPER, H. W. .....543 Franklin St., Op.
EDSON, R. A. .....957 Delaware Ave., Op.
FAIRBAIRN, JOHN F. .....925 Delaware Ave., ALR
FLAGG, J. D. .....158 Anderson Place, Op. A
GLOSSER, HERBERT H. .....448 Franklin St., Op.
HEALY, JOHN F. .....925 Delaware Ave., ALR
KOENIG, IVAN J. .....40 North St., Op.
LE WIN, THURBER .....112 Linwood Ave., Op.
LEWIS, F. PARK .....454 Franklin St., Op.
LUHR, A. F. .....40 W. North St., Op.
MCKEE, OTTO S. .....83 Bryant St., ALR
MARCH, CLARA A. .....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON .....131 Linwood Ave., Op.
RENNER, W. S. .....341 Linwood Ave., ALR

RIWCHUN, MEYER H. .....468 Delaware Ave., Op.
TROTTER, HOMER A. .....40 North St., ALR
WEED, HARRY M. .....196 Linwood Ave., Op.
WURTZ, W. J. M. .....333 Linwood Ave., ALR

CORTLAND

HIGGINS, R. P. .....20 Court St., OALR
WHITE, O. E. .....28 No. Main St., ALR

DUNKIRK

JACKLE, ARTHUR F. .....63 E. 4th St., Op.

ELMIRA

GLOVER, ARTHUR C. .....311 W. Church St., OALR

FLUSHING

GOODE, EDWARD ALLEN .....143-28 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR .....423 S. Main St., Op.

GLENS FALLS

BUCK, CLAIR A. .....191 Glen St., ALR
FIELDING, F. G. .....70 Ridge St., ALR

GOVERNOR'S ISLAND

SIMMONS, RALPH H. (Major M.C.)
2nd Corps Area Hosp., Fort Jay, ALR

JAMAICA

BENDER, M. S. .....88-05 150th St., ALR
KEELER, LANSING H. .....89-04 148th St., ALR
MILLER, SAUL .....8805 150th St., Op.

JAMESTOWN

REGER, H. S. .....Physicians Bldg., OALR

LITTLE FALLS

NEWTON, MORRIS H. .....Burrell Bldg., Op.

LOCKPORT

PARKER, HARRY L. .....88 East Ave., ALR
RINGUEBERG, EUGENE N. S. .....13 Main St., Op.

LONG ISLAND — HEMPSTEAD

HUGHES, WENDELL L. .....Professional Bldg., Op.
HULKA, J. H. .....30-28 36th St., OALR

MIDDLETOWN

HICKS, WM. J. .....15 King St., ALR
NELLER, WALTER I. .....121 Wickham Ave., Op.

MT. VERNON

KEEFE, WM. JOHN .....110 E. Sidney Ave., ALR
THOMSON, J. J. .....531 E. Lincoln Ave., ALR

NEW ROCHELLE

BECK, A. L. .....421 Huguenot St., OALR
HETRICK, L. E. .....260 North Ave., ALR
WOODRUFF, WALTER STUART.....421 Huguenot St., ALR

NEW YORK CITY

AEBLI, RUDOLF .....30 E. 40th St., Op.
ALMOUR, RALPH .....51 W. 73rd St., ALR
AUERBACH, JULIUS .....1016 Fifth Ave., ALR
BALLIN, M. J. .....20 E. 76th St., ALR
BASSEN, ED. J. .....698 Madison Ave., Op.
BEACH, B. S. .....140 W. 86th St., Op.
BENDER, M. S. .....30 E. 40th St., ALR
BERENS, CONRAD .....35 E. 70th St., Op.
BERKE, RAYNOLD N. .....30 W. 59th St., Op.
BERLINER, M. L. .....57 W. 57th St., Op.
BOYES, T. L. .....100 W. 59th St., Op.
BRANIGAN, CHAS. H. .....399 Park Ave., Op.
BREUNING, PAUL H. .....30 W. 59th St., ALR
BRUCE, G. M. .....635 W. 165th St., Op.
BRUDER, JOSEPH .....57 W. 57th St., OALR
BULLWINKEL, H. GRIFFIN .....30 E. 76th St., ALR
CARTER, W. W. .....140 E. 54th St., ALR
CASTROVIEJO, RAMON .....635 W. 165th St., Op.
COHEN, MARTIN .....29 E. 64th St., Op.
COHN, FELIX .....945 West End Ave., ALR
COLBERT, ROBERT MARTIN .....57 W. 57th St., ALR
CORYA, HAROLD W. .....30 E. 40th St., ALR
COTT, D. N. .....25 Central Ave., St. George, Staten Isl., ALR
CUNNING, DANIEL S. .....145 E. 54th St., OALR
DAVIDSON, MORRIS .....80 Centre St., Dept. of Labor, Op.
DAVIS, A. EDWARD .....40 E. 61st St., Op.
DAVIS, G. E. .....1060 Fifth Ave., OALR
DENISON, WARD C. .....20 E. 53rd St., ALR
DEPPING, CHAS. WM. .....17 W. 54th St., ALR
DIXON, G. S. .....1150 Fifth Ave., R
DLUGASCH, LOUIS .....110 E. 36th St., ALR
DOHERTY, W. B. .....150 W. 55th St., Op.
DUNNINGTON, J. H. .....30 W. 59th St., Op.
ELLIS, Z. H. .....58 E. 65th St., Op.
FERNLUND, GEORGE BYRON ....8577 112th St., Richmond Hill, OALR
FITZGERALD, FRED J. C. .....424 Madison Ave., Op.
FOWLER, ROBERT HENRY .....153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON .....30 W. 59th St., ALR
FRANKEL, EDWARD, JR. .....217 E. 17th S., OAL
FREY, GUERNSEY .....121 E. 60th St., Op.

FRIEDMAN, BENJAMIN .....6 W. 77th St., Op.
GATEWOOD, W. L. .....330 Park Ave., ALR
GERSTLEY, MANFRED JOS. .....30 E. 40th St., Op.
GIORDANO, A. J. .....749 E. 183rd St., ALR
GOLDMAN, A. MILTON .....121 E. 60th St., Op.
GOLDMAN, H. G. .....57 W. 58th St., ALR
GOLDSTEIN, ISADORE .....1095 Park Ave., Op.
GOTTLIEB, MARK J. .....2 W. 87th St., ALR
GRAEBNER, HERBERT .....45 Park Ave., ALR
GRESSER, EDWARD BELLAMY .....39 Fifth Ave., Op.
GRUSHLOW, ISRAEL .....271 Central Park, W., ALR
GUY, LOREN P. .....116 E. 62nd St., Op.
HALLETT, G. DEWAYNE .....274 W. 86th St., Op.
HARDY, LEGRAND H. .....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON .....104 E. 40th St., ALR
HARTSHORNE, ISAAC .....30 W. 59th St., Op.
HASKIN, W. H. .....300 Park Ave., ALR
HAYS, HAROLD M. .....133 E. 58th St., ALR
HETRICK, L. E. .....40 E. 61st St., ALR
HIRSCH, CAESAR .....667 Madison Ave., ALR
HORN, WALTER L. .....1040 Park Ave., ALR
HURD, L. M. .....39 E. 50th St., ALR
HUGHES, WENDEL LOCHEAD .....20 E. 53rd St., Op.
HUNT, WESTLEY MARSHALL .....33 E. 68th St., ALR
HUTCHINSON, A. T. .....30 W. 59th St., ALR
IMPERATORI, C. J. .....108 E. 38th St., ALR
INGERMAN, S. M. .....418 Central Park, W., OALR
JACOBS, S. M. .....1749 Grand Concourse, OALR
JARECKY, HERMAN .....168 W. 86th St., OALR
JEROME, WM. Z. .....436 Fort Washington Ave., ALR
JOHNSON, THOMAS HOLLAND .....30 W. 59th St., Op.
JONES, MARVIN F. .....121 E. 60th St., ALR
JOSEPHSON, EMANUEL M. .....993 Park Ave., ALR
JUDD, H. B. .....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC .....140 E. 54th St., OALR
KEND, LEE .....145 Central Park West, ALR
KEY, BEN WITT .....100 W. 59th St., Op.
KING, JAMES J. .....133 E. 58th St., ALR
KIRBY, D. B. .....780 Park Ave., Op.
KNAPP, ARNOLD .....10 E. 54th St., Op.
KNAPP, ARTHUR A. .....133 E. 58th St., Op.
KNIGHTON, W. S. .....40 E. 61st St., Op.
KOLLER, CARL .....30 E. 58th St., Op.
KOPETZKY, S. J. .....51 W. 73rd St., ALR
KRUG, ERNEST F. .....988 Fifth Ave., Op.
LAMBERT, ROBERT K. .....1010 Fifth Ave., Op.
LA VIGNE, A. A. .....471 Park Ave., OALR
LEVINE, JOSEPH .....105 E. 63rd St., Op.
LITTWIN, CHAS. .....550 Park Ave., Op.
LOEWE, WALTER R. .....1114 Madison Ave., Op.

LUBMAN, MAX ..... 118 E. 93rd St., ALR
MCAULIFFE, GERVAIS W. ..... 110 W. 55th St., ALR
MCDANNALD, C. E. ..... 100 W. 59th St., Op.
MCDONALD, DENNIS J. ..... 17 E. 38th St., ALR
MCKEOWN, HUGH S. ..... 30 W. 59th St., Op.
MAY, CHAS. H. ..... 698 Madison Ave., Op.
MAYBAUM, J. L. ..... 1070 Park Ave., ALR
MEEK, R. E. ..... 729 Park Ave., Op.
MEIERHOF, E. L. (Retired) ..... care Guaranty Trust Co., OALR
MILLER, JOSEPH WILLIAM ..... 305 E. 17th St., ALR
MINSKY, HENRY ..... 1 W. 86th St., Op.
MITTENDORF, A. D. ..... 399 Park Ave., Op.
MITTENDORF, W. KEITH ..... 115 E. 53rd St., Op.
MORRISON, WALLACE ..... 39 E. 50th St., ALR
MOYER, JOHN SANFORD ..... 239 E. 75th St., ALR
MOYLE, EUGENE H. ..... 1088 Park Ave., ALR
MYERSON, M. C. ..... 136 E. 64th St., ALR
NEAL, PHIL HAWKINS ..... 135 E. 65th St., ALR
NISSELSON, MAX ..... 1018 E. 163rd St., ALR
NORTHCOTT, THOMAS A. ..... 17 E. 38th St., Op.
OAST, SAMUEL P., JR. ..... 30 E. 40th St., Op.
PALMER, ARTHUR ..... 667 Madison Ave., ALR
PAYNE, B. F. ..... 35 E. 70th St., Op.
PETERSON, R. A. ..... 150 Fifth Ave., OALR
REESE, ALGERNON B. ..... 73 E. 71st St., Op.
ROSENBAUM, MORRIS ..... 61 W. 88th St., Op.
ROSENBLUTH, MICHAEL ..... 1018 E. 163rd St., ALR
SACKIN, DAVID ..... "St. Moritz," 50 Central Park South, ALR
SAMUELS, BERNARD ..... 57 W. 57th St., Op.
SCAL, JOSEPH COLEMAN ..... 44 W. 74th St., ALR
SCHEER, HENRY M. ..... 522 West End Ave., ALR
SCHILLER, A. NOAH ..... 146 Central Park West, ALR
SCHLIVEK, KAUFMAN ..... 1016 Fifth Ave., Op.
SCHMIDT, OTTO V. M. ..... 111 E. 61st St., ALR
SCHOENBERG, MARK J. ..... 1160 Park Ave., Op.
SCHUGT, HENRY P. ..... 30 E. 40th St., ALR
SCHWARTZ, A. A. ..... 7 W. 71st St., ALR
SETTEL, NATHAN ..... 10 W. 82nd St., ALR
SEWARD, J. A. ..... 115 E. 61st St., ALR
SITCHEVSKA, OLGA L. ..... 160 W. 87th St., Op.
SMITH, JAS. M. ..... 123 E. 53rd St., ALR
SOMBERG, J. S. ..... 470 Park Ave., Op.
STARK, JESSE B. ..... 45 Park Ave., Op.
STRAATSMA, CLARENCE R. ..... 899 Park Ave., ALR
STURGES, LEIGH F. ..... 121 E. 60th St., ALR
TUSAK, E. A. ..... 115 E. 90th St., Op.
WACHSBERGER, ALFRED ..... 51 W. 73rd St., ALR
WEEKS, WEBB WILLIAM ..... 20 E. 53rd St., Op.
WEXLER, DAVID ..... 179 E. 79th St., Op.

WHEELER, J. M. .....635 W. 165th St., Op.
WHELAN, WILLIAM F. .....2015 Walnut St., ALR
WHITE, JAMES W. .....15 Park Ave., Op.
WIENER, ALFRED .....550 Park Ave., OALR
WINCOR, HENRY G. .....2021 Grand Concourse, Op.
WOODMAN, DEGRAAF .....156 E. 37th St., ALR

NIAGARA FALLS

PRICE, NORMAN W. .....445 Third St., OALR
ROOKER, A. M. .....203 Gluck Bldg., ALR

NYACK

TOMS, S. W. S. .....120 S. Broadway, OALR

ONEIDA

CROCKETT, R. L. .....302 Main St., Op.

ONEONTA

HALL, EDWIN P. .....76 Chestnut St., ALR

PLATTSBURG

SCHNEIDER, ANTON S. .....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A. .....103 Hooker Ave., OALR

ROCHESTER

AVERY, ELROY J. .....11 N. Goodman St., ALR
BARBER, F. .....75 S. Fitzhugh St., Op.
BARLOW, ROY A. .....Medical Arts Bldg., 277 Alexander St., ALR
BISHOP, VERNON L. .....277 Alexander St., ALR
CARROLL, GEORGE G. .....5 North Goodman St., OALR
CLARK, L. H. .....337 Monroe Ave., OALR
GIPNER, J. F. .....Medical Arts Bldg., Op.
JONES, L. W. .....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON .....Gas and Electric Bldg., Op.
LERNER, MACY LEVI .....332 Park Ave., Op.
MCDOWELL, N. D. .....275 Alexander St., OALR
MORRIS, A. G. .....Medical Arts Bldg., ALR
NASH, C. STEWART .....277 Alexander St., ALR
SHAPERO, I. M. .....289 Alexander St., OALR
SNELL, ALBERT C. .....53 S. Fitzhugh St., Op.
STOKES, HARRY B. .....26 S. Goodman St., ALR

ROME

VALONE, FRANK H. .....112 E. Dominick St., ALR

RYE

SOMBERG, J. S. .....127 Overdale Road, Greenhaven, Op.

SYRACUSE

BRITTEN, G. S. ..... Medical Arts Bldg., ALR
BROWN, MORTIMER G. ..... 713 E. Genesee, ALR
BRUST, H. O. ..... 820 S. Crouse Ave., ALR
CAVE, LINUS S. ..... 221 Physicians Bldg., ALR
FOWLER, S. R. ..... University Block, ALR
GILLETTE, D. F. ..... State Tower Bldg., Op.
HALL, EDWIN P. ..... State Tower Bldg., ALR
HOOPLE, GORDON D. ..... Medical Arts Bldg., ALR
JOY, HAROLD HENRY ..... 504 State Tower Bldg., Op.
KAUFFMAN, A. B. ..... Chimes Bldg., ALR
KLINE, H. G. ..... State Tower Bldg., ALR
LEVY, HARRY H. ..... State Tower Bldg., Op.
MARLOW, SEARLE B. ..... 1004 State Tower Bldg., Op.
MOORE, ROY S. ..... Medical Arts Bldg., ALR
O'CONNOR, FREDERICK J. ..... 713 E. Genesee St., ALR
RUBENSTEIN, A. H. ..... Medical Arts Bldg., ALR
WEBSTER, F. R. ..... 720 State Tower Bldg., Op.

TARRYTOWN

NEWLANDS, W. ALEXANDER ..... 19 S. Broadway, ALR

TROY

SULZMAN, FRANK MALCOLM ..... 1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H. ..... 258 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S. ..... 168 Sterling St., Op.
HENDERSON, L. E. ..... 168 Sterling St., Op.
MCASKILL, JAS. E. ..... Woolworth Bldg., ALR

WHITE PLAINS

MORIARTY, RICHARD W. ..... Medical Center, OALR

WOODHAVEN

FROTHINGHAM, EDWARD ..... 8644 94th St., OALR

YONKERS

WOLCOTT, CHAS. C. ..... 27 Ludlow St., ALR

NORTH CAROLINA

ASHEVILLE

GREENE, JOSEPH BERRY ..... Haywood Bldg., ALR
SWANN, CECIL C. ..... 706 Flatiron Bldg., ALR

CHARLOTTE

MATHESON, J. P. ..... 106 W. 7th St., ALR
PEELER, C. N. ..... 106 W. 7th St., OALR
SLOAN, HENRY L. ..... 106 W. 7th St., Op.
WHISNANT, A. M. ..... 517 Professional Bldg., OALR

GREENSBORO

REAVES, W. P. .....Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE .....Medical Arts Bldg., ALR

MOREHEAD CITY

JOHNSON, OTIS H. .....OALR

NEW BERN

DANIELS, R. L. .....Box 685, OALR

RALEIGH

HICKS, VONNIE MONROE .....Professional Bldg., Op.
WRIGHT, J. B. .....Professional Bldg., ALR

WINSTON-SALEM

DAVIS, THOMAS W. .....210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

BUCKINGHAM, TRACY W. .....405 Broadway, ALR
CONSTANS, GEORGE MAURICE .....Clinic Bdlg., Op.

FARGO

LARSON, G. ARTHUR .....A.O.U.W. Bldg., Op.
RINDLAUB, ELIZABETH P. .....de Lendrecie Bldg., OALR
RINDLAUB, JOHN H. .....de Lendrecie Bldg., OALR

MINOT

MCCANNEL, A. D. .....15A Central Ave. W., OALR

OHIO

AKRON

ARBUCKLE, DAVID S. .....513 W. Market St., ALR
BROWN, L. E. .....Second Nat'l Bank Bldg., OALR
BRUMBAUGH, JOHN D. .....Second Nat'l Bank Bldg., ALR
MOORE, T. K. .....Second Nat'l Bank Bldg., OALR
SEIDEL, U. D. .....Second Nat'l Bank Bldg., ALR

ALLIANCE

KING, GEORGE L., JR. .....537 E. Market St., Op.
KING, G. L., SR. .....537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. .....112 W. Columbus Ave., OALR
HARBERT, JOHN P. .....135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. ..... 309 Brant Bldg., ALR
TOOT, JOSEPH F. ..... 923 First Nat'l Bank Bldg., Op.
WEAVER, HOMER V. ..... 228 Cleveland Ave., N.W., ALR

CINCINNATI

ABRAHAMSON, IRA R. ..... 925 Fifth Third Union Trust Bldg., Op.
ASBURY, MARY K. ..... 2466 Grandin Road, Op.
BEYER, A. G. ..... Doctors Bldg., OALR
BROWN, ALBERT LOUIS ..... Doctors Bldg., Op.
BRYANT, BEN L. ..... 19 Garfield Place, ALR
FISCHBACH, VICTOR W. ..... Union Central Bldg., ALR
GOODYEAR, H. M. ..... Doctors Bldg., ALR
HANS, C. L. ..... Provident Bank Bldg., OALR
HARRIS, W. C. ..... Groton Bldg., ALR
HENDICKS, LOUIS J. ..... 210 W. 12th St., Op.
HOFLING, CHARLES A. ..... 707 Race St., Op.
HOWARD, ROBERT E. ..... Union Central Bldg., ALR
IGLAUER, SAMUEL ..... 707 Race St., ALR
JONES, C. C. ..... Doctors Bldg., ALR
KING, CLARENCE ..... Union Central Bldg., Op.
KING, ED. ..... 1002 Carew Tower, ALR
LYLE, DONALD J. ..... 909 Carew Tower, Op.
MITHOEFER, W. ..... Doctors Bldg., ALR
PHINNEY, F. D. ..... 22 W. 7th St., OALR
RAY, VICTOR, JR. ..... Carew Tower, Op.
RAY, VICTOR, SR. ..... Carew Tower, Op.
REID, H. W. ..... Doctors Bldg., Op.
SELTZ, SAMUEL ..... Provident Bank Bldg., ALR
SIEGEL, FRANCIS X. ..... 610 Union Central Life Bldg., OALR
STANBERY, HENRY ..... 566 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., ALR
STOLL, KARL LUDWIG ..... 19 West 7th St., OALR
TANGEMAN, H. F. ..... 606 Union Central Life Bldg., ALR
THOMPSON, E. H. ..... 8 W. 9th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR
VAIL, D. T., JR. ..... Carew Tower, 441 Vine St., Op.
VAIL, H. H. ..... Carew Tower, 441 Vine St., ALR

CLEVELAND

ABBOTT, W. J. ..... Carnegie Medical Bldg., OALR
BRUNER, ABRAM BRENEMAN ..... 1214 Guardian Bldg., OALR
BRUNER, WM. E. ..... Guardian Bldg., Op.
BUCHER, WALTER M. ..... 4187 Pearl Road, ALR
BURKE, T. A. ..... Rose Bldg., OALR
CHAMBERLAIN, WEBB PARKS ..... 7405 Detroit Ave., Op.
CUTLER, FRANKLIN E. ..... Hanna Bldg., ALR
DIXON, FRED WILLIS ..... 1029 Rose Bldg., ALR

ENGLER, CLARENCE WM. ..... 2323 Prospect Ave., ALR
FORSYTHE, S. T. ..... Rose Bldg., OALR
JACOBY, MARK W. ..... Hanna Bldg., Op.
KENDALL, M. RAYMOND ..... Hanna Bldg., ALR
KINNEY, CHARLES ED. ..... 10515 Carnegie Ave., ALR
LARGE, S. H. ..... Hanna Bldg., ALR
MCDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... Hanna Bldg., Op.
METZENBAUM, MYRON ..... Carnegie Medical Bldg., ALR
MITCHELL, HORACE E. ..... 14805 Detroit Ave., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Medical Arts Bldg., Op.
MOTTO, MICHAEL PAUL ..... Rose Bldg., Op.
MULLIN, WM. V. ..... 2020 E. 93rd St., ALR
MUSSUN, W. G. ..... Medical Arts Bldg., ALR
NELSON, CHAS. F. ..... 606 Schofield Bldg., OALR
PITKIN, C. E. ..... Carnegie Medical Bldg., ALR
PRENDERGAST, D. A. ..... Medical Arts Bldg., OALR
PUTNAM, F. I. ..... 1401 Prospect Ave., OALR
QUITTNER, S. S. ..... 10515 Carnegie Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... 10515 Carnegie Ave., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
RUST, E. G. ..... 1422 Euclid Ave., Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TUCKERMANN, W. C. ..... 733 Osborn Bldg., Op.
TUCKERMANN, W. H. ..... 733 Osborn Bldg., OALR
WAUGH, J. M. ..... Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. ..... 10515 Carnegie Ave., ALR

COLUMBUS

BEATTY, H. G. ..... 681 E. Broad St., ALR
BROWN, JOHN E. ..... 370 E. Town St., OALR
CLARK, I. G. ..... 188 E. State St., OALR
DAVIES, MORGAN C. ..... 269 E. State St., Op.
DAVIS, WM. C. ..... 327 E. State St., Op.
FROST, ALBERT DALBEY ..... 681 E. Broad St., Op.
HARRIS, ED. WM. ..... 681 E. Broad St., ALR
HAUER, A. M. ..... 327 E. State St., ALR
HELFRICH, ED. D. ..... 269 E. State St., ALR
JOHNSON, DAVID M. ..... 327 E. State St., ALR
MEANS, R. G. ..... 40 S. Third St., ALR
PERRY, CLAUDE S. ..... 40 S. Third St., Op.
POSTLE, C. D. ..... 240 E. State St., ALR
PROUT, A. W. ..... 380 E. Town St., OALR
SANOR, DANIEL G., JR. ..... 206 E. State St., ALR
SILBERNAGEL, C. E. ..... 247 E. State St., OALR

STOUTENBOROUGH, WM. A. .....21 E. State St., OALR
SULZER, G. A. .....327 E. State St., OALR
THOMAS, FRANCIS W. .....327 E. State St., OALR
TIMBERMAN, ANDREW .....Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S. .....Fidelity Bldg., OALR
BLACKBURN, W. J. .....663 Reibold Bldg., OALR
CULLER, ARTHUR M. .....Fidelity Medical Bldg., Op.
DRISCOLL, FREDERICK JAS. .....Reibold Bldg., ALR
DUTROW, H. V. .....1040 Fidelity Medical Bldg., OALR
FARMER, A. G. .....Fidelity Medical Bldg., OALR
FOUTS, JOHN D. .....Fidelity Medical Bldg., OALR
HARRIS, H. B. .....Fidelity Medical Bldg., OALR
KILBOURNE, P. H. .....Fidelity Medical Bldg., OALR
MILLETTE, J. W. .....Reibold Bldg., OALR

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STUEBER, PAUL J. .....209 W. North St., OALR

LORAIN

MONOSMITH, O. B. .....O. P. S. Bldg., OALR

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WISE, RALPH C. .....News Bldg., OALR

MARIETTA

SAUER, W. W. .....231 Third St., OALR

MARION

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SCOTT, MERLE E. .....First Nat'l Bank Bldg., OALR

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WILLIAMS, WALTON H. .....Castell Bldg., OALR

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HATCH, C. B. .....3 W. Church St., OALR

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KING, LOWELL W. .....470 E. State St., ALR

SANDUSKY

KRAMER, JULIUS C. ..... 130 E. Market St., ALR

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HARTLEY, F. A. ..... 302 E. High St., OALR
HOGUE, D. W. ..... 34 W. High St., OALR
MINOR, C. L. ..... First Nat'l Bank Bldg., OALR

STEUBENVILLE

GOURLEY, G. F. ..... Sinclair Bldg., OALR

TIFFIN

PORTER, E. H. ..... 85 Madison St., OALR

TOLEDO

ALDERDYCE, WM. W. ..... 513 Madison Ave., OALR
KING, CHAS. R. ..... Spitzer Bldg., OALR
LUKENS, CHARLES ..... 316 Michigan St., OALR
LUKENS, JOHN A. ..... 316 Michigan St., ALR
NIPPE, LEONARD ..... 316 Michigan St., OALR
SNYDER, W. H. ..... 2001 Colingwood Ave., OALR
STEINFELD, A. L. ..... 125 Fifteenth St., OALR
WINGER, IRA B. ..... 225 Michigan St., OALR

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STIMSON, GEORGE W. ..... Packard Bldg., OALR

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SHIELDS, LAWRENCE ..... Allen Bldg., ALR

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EVANS, WILLIAM H. ..... 24 Wick Ave., ALR
GOLDCAMP, ED. C. ..... Dollar Bank Bldg., ALR
HARTZELL, SOL. M. ..... Dollar Bank Bldg., OALR
KEYS, JOHN E. L. ..... 613 Home Savings and Loan Bldg., Op.

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ENID

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HUBER, WALTER A. ..... Medical Arts Bldg., Op.
ROTH, ALBERT W. ..... Medical Arts Bldg., OALR
SMITH, RURIC N. ..... Medical Arts Bldg., ALR

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BAKER

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KISTNER, F. B. ..... 600 Mayer Bldg., OALR
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CLEMENT, L. O. ..... Guardian Bldg., OALR

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GLOVER, S. P. ..... 1118 Twelfth Ave., OALR

ARDMORE

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SCROGGS, J. J. ..... 218 E. Third St., Op. A

BETHLEHEM

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DOANE, L. L. ..... 357 N. Main St., OALR
HAZLETT, L. R. ..... Savings and Trust Bldg., OALR

CHESTER

CROSS, G. H. ..... 525 Welsh St., Op.
ORR, ADRIAN V. B. ..... 525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H. ..... 308 First Nat'l Bank Bldg., OALR

DANVILLE

NEBINGER, REID ..... Geisinger Memorial Hospital, ALR

DU BOIS

BEALS, C. WEARNE ..... 41 N. Brady St., ALR

EASTON

BLOOM, C. HUGH ..... 65 N. Third St., ALR

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FERMAN, JOHN W. ..... OALR

ERIE

DELANEY, JAS. H. ..... 138 W. 9th St., Op.
DENNIS, EDWARD ..... 309 W. 10th St., OALR
FUST, JOHN H. E. ..... 231 W. 7th St., OALR
JACKSON, J. D. ..... 232 W. 8th St., OALR
RUSSELL, J. A. MERLE ..... 813 Sassafras St., OALR
SCHLINDWEIN, G. WM. ..... 138 W. 9th St., OALR
SHREVE, OWEN M. ..... 162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B. ..... West Park and Liberty St., OALR

GREENSBURG

FAIRING, JOHN W. ..... Tribune Review Bldg., OALR
MCKEE, CLAUD W. ..... Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L. ..... 618 N. 3rd St., OALR
FARNSLER, H. HERSHEY ..... 1438 Market St., OALR
GRACEY, GEORGE F. ..... 209 State St., OALR

HAZLETON

REICHE, O. C. ..... Markle Bank Bldg., Op.

HUNTINGDON

HUNT, WILLIAM T., JR. ..... 514 Penn St., ALR

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BARKER, O. G. A. ..... 804 Johnstown Trust Bldg., Op.
GRIFFITH, HAROLD M. ..... 804 Johnstown Trust Bldg., Op.
HARRIS, C. M. ..... 604 Johnstown Trust Bldg., OALR
RUSH, C. C. ..... 907 U. S. Nat'l Bank Bldg., OALR

MIDDLETOWN

GEORGE, HENRY W. ..... 19 N. Union St., Op.

NEW CASTLE

JAQUISH, CHAS. J. ..... 211 E. Wallace Ave,. ALR

NEW KENSINGTON

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STEIM, J. M. ..... First Nat'l Bank Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH .... Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE ..... 824 De Kalb St., ALR

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ADLER, FRANCIS HEED ..... 313 S. 17th St., Op.
ALEXANDER, GEORGE J. ..... 1930 Chestnut St., ALR
ANNON, W. T. ..... 235 S. 15th St., ALR
APPLEMAN, L. F. ..... 308 S. 16th St., Op.
BABBITT, JAMES A. ..... 1912 Spruce St., ALR
BAER, BENJAMIN F., JR. ..... 1927 Spruce St., Op.
BALDWIN, KATE W. ..... 1117 Spruce St., ALR
BALENTINE, P. L. ..... 1524 Chestnut St., Op.
BATSON, OSCAR V. ..... 3502 Hamilton St., ALR
BLAIR, MORTIMER W. ..... N.E. Cor. 20th and Chestnut St., Op.
BOEHRINGER, H. WINFIELD ..... 700 Central Medical Bldg., OALR

BRICKER, SACKS .....3701 N. Broad St., ALR
BROWN, SAMUEL HORTON .....1930 Chestnut St., Op.
BRUMM, SETH A. .....Medical Arts Bldg., OALR
BURNS, L. J. .....1930 Chestnut St., ALR
BUTT, MIRIAM M. .....2045 Chestnut St., Op.
CARPENTER, C. .....1930 Chestnut St., Op.
CLERF, LOUIS HENRY .....1530 Locust St., ALR
COATES, GEORGE M. .....1721 Pine St., ALR
COHEN, HERMAN B. .....The Lenox, Spruce and 13th Sts., ALR
COHEN, SAMUEL .....2009 Pine St., ALR
COWAN, ALFRED .....1930 Chestnut St., Op.
CREIGHTON, WM. J. .....258 S. 18th St., Op.
DAVIS, J. LESLIE .....135 S. 18th St., ALR
DAVIS, T. CARROLL .....3128 N. Broad St., ALR
DAVIS, WARREN B. .....135 S. 18th St., ALR
DEICHLER, LYN W. .....1930 Chestnut St., OALR
DINTENFASS, HENRY .....1305 Spruce St., ALR
EMBERY, FRANK .....4801 Penn St. (Frankford) ALR
ERSNER, MATTHEW S. .....1915 Spruce St., ALR
EVES, C. C. .....1910 Spruce St., ALR
FEWELL, A. G. .....1924 Pine St., Op.
FRY, W. E. .....1912 Spruce St., Op.
GLEASON, E. B. .....2112 Pine St., ALR
GODDARD, H. M. .....1531 Spruce St., ALR
GORDON, WM. .....1721 Pine St., ALR
GOUTERMAN, JOSEPH I. .....1930 Chestnut St., Op.
GOWEN, FRANCIS V. .....904 E. Chelton Ave., ALR
GRIMES, CHAS. H. .....3546 N. Broad St., ALR
GRISCOM, J. MILTON .....2213 Walnut St., Op.
HAWMAN, ERLE G. .....4051 N. Broad St., Op.
HEED, C. R. .....1205 Spruce St., Op.
HEWSON, WM. .....6013 Greene St., Germantown, ALR
HIRST, OTTO C. .....255 S. 17th St., ALR
HOLLOWAY, THOMAS B. .....1819 Chestnut St., Op.
HOUSER, KARL M. .....2010 Spruce St., ALR
HUNTER, R. J. .....2011 Chestnut St., ALR
HUSIK, D. N. .....1930 Chestnut St., ALR
IVY, ROBERT H. .....1930 Chestnut St., ALR
JACKSON, CHEVALIER .....3432 N. Broad St., L
JACKSON, CHEVALIER L. .....3432 N. Broad St., L
KAUFMAN, A. SPENCER .....1923 Spruce St., ALR
KEELER, J. C. .....Medical Arts Bldg., ALR
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LEWIS, FIELDING O. .....259 S. 17th St., ALR

LILLIE, WALTER I. .....3701 N. Broad St., Op.
LOFTUS, JOHN EDWARD .....1930 Chestnut St., ALR
LUKENS, ROBERT McD. .....1923 Spruce St., ALR
LUONGO, R. A. .....1713 Pine St., ALR
McANDREWS, LEO F. .....1930 Chestnut St., Op.
MACKENZIE, GEORGE W. .....269 S. 19th St., OALR
MANN, BENJAMIN H. ....N.E. Corner 20th and Chestnut Sts., Op.
MENDEL, JAS. H. .....Germantown Prof. Bldg., ALR
MILLER, M. VALENTINE....114 W. Phil-Ellena St., Mt. Airy, ALR
MITCHELL, E. K. .....710 W. Lehigh Ave., ALR
MOORE, WILLIAM FREDERICK .....255 S. 16th St., ALR
O'BRIEN, THOMAS A. .....1727 W. Girard Ave., Op.
OLSHO, S. L. .....235 S. 15th St., OALR
PERSKY, ABRAM H. .....1922 Spruce St., ALR
PETER, L. C. .....1930 Chestnut St., Op.
PONTIUS, P. J. .....1831 Chestnut St., Op.
REESE, WARREN S. .....230 S. 21st St., Op.
RIDPATH, ROBERT F. .....N.E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C. .....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON .....1924 Pine St., Op.
SARTAIN, PAUL J. .....2006 Walnut St., Op.
SCARLETT, HUNTER W. .....230 S. 21st St., Op.
SCHWEINITZ, GEORGE E. DE .....1705 Walnut St., Op.
SENER, WALTER J. .....Physicians Bldg., Op.
SHANNON, C. E. G. .....1930 Chestnut St., Op.
SHUSTER, BENJAMIN HARRISON .....1930 Chestnut St., ALR
SKILLERN, S. R., JR. .....1734 Pine St., ALR
SLOANE, HENRY O. .....1717 Pine St., Op.
SLOTKIN, HERMAN B. .....1912 Pine St., ALR
SPAETH, EDMUND B. .....1930 Chestnut St., Op.
SPENCER, W. H. .....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M. .....1831 Chestnut St., Op.
STOUT, PHILIP S. .....269 S. 19th St., ALR
TASSMAN, ISAAC S. .....Medical Arts Bldg., Op.
TOWSON, CHAS. E.
Germantown Prof. Bldg., Greene and Coulter Sts., ALR
TUCKER, GABRIEL .....326 S. 19th St., B
WAGERS, ARTHUR J. .....1429 Spruce St., ALR
WEINSTEIN, M. A. .....1309 Spruce St., ALR
WHELAN, G. L. .....2100 Walnut St., ALR
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PITTSBURGH

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BLAIR, W. W. .....121 University Pl., Op.
CARSON, W. E. .....Jenkins Arcade Bldg., Op.
CONWAY, JOHN M. .....5151 Jenkins Arcade, ALR

COOPER, ROY C. ..... 510 S. Aiken Ave., ALR
CURRY, GLENDON E. ..... Westinghouse Bldg., Op.
DAY, KENNETH M. ..... 121 University Pl., ALR
DAY, EWING W. ..... 121 University Pl., OALR
FISCHER, N. ARTHUR ..... 623 Union Trust Bldg., ALR
GROSS, JULIUS E. ..... 3401 Fifth Ave., ALR
GUERINOT, A. J. ..... 604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A. ..... Highland Bldg., ALR
HAMPSEY, A. R. ..... 806 May Bldg., ALR
HECKEL, EDWARD B. ..... 500 Penn. Ave., Op.
JENNINGS, CHAS. W. ..... Hiland Bldg., Op.
KNEEDLER, G. C. ..... Clark Bldg., ALR
KREBS, ADOLPH ..... Jenkins Arcade Bldg., Op.
LINN, J. G. ..... Jenkins Arcade Bldg., Op.
MCCOLLOUGH, THOMAS B. ..... 121 University Pl., ALR
MCCREADY, J. HOMER ..... 121 University Pl., ALR
MCKEE, GEORGE J. ..... Westinghouse Bldg., ALR
MACLACHLIN, A. A. ..... May Bldg., ALR
MARKEL, J. CLYDE ..... Westinghouse Bldg., Op.
MARSHALL, WATSON ..... 121 University Pl., ALR
MEANOR, WM. C. ..... 619 Westinghouse Bldg., Op.
PATTERSON, ELLEN J. ..... 121 University Pl., ALR
PLUMER, JOHN S. ..... 121 University Pl., Op.
REED, CHAS. L. ..... Jenkins Bldg., Op.
SEEGMAN, SIMON ..... Medical Bldg., ALR
SHUMAN, G. H. ..... Park Bldg., Op.
SIMPSON, JOHN REID ..... Medical Arts Bldg., ALR
SMITH, STANLEY S. ..... 121 University Pl., Op.
STIEREN, E. ..... 626 Union Trust Bldg., Op.
TAYLOR, M. C. ..... May Bldg., Op.
THORPE, H. E. ..... 8075 Jenkins Arcade Bldg., Op.
TODD, GROVER CLEVELAND ..... Jenkins Arcade Bldg., ALR
TURNER, H. H. ..... Clark Bldg., Op.
WEISSER, E. A. ..... 806 May Bldg., Op.
YOST, WALTER MILTON ..... Westinghouse Bldg., Op.

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READING

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HIGH, ISAAC B. ..... 326 N. 5th St., ALR
LANDIS, JAMES E. ..... 517 Walnut St., ALR
RHODE, SOLON L. ..... 238 N. 6th St., OALR
STOCKLER, J. A. ..... 332 N. 5th St., OALR

RIDGWAY

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CURTIN, EUGENE A. .....506 Spruce, OALR
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MCMURRAY, J. B. .....Washington Trust Bldg., OALR

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WILKES-BARRE

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CARR, GEORGE W. .....54 S. Franklin St., OALR
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MCLAUGHLIN, P. A. .....71 S. Washington St., OALR
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SCHAPPERT, N. LOUIS .....59 S. Washington St., OALR

WILLIAMSPORT

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KLINEDINST, JOHN FRED .....220 S. George St., Op. A

PUERTO RICO

SAN JUAN

FONT, JUAN H.....Cruz St. No. 13, ALR

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BOLOTOW, NATHAN A. ..... 108 Waterman St., ALR
CAPRON, F. P. ..... 118 Angell St., OALR
DIMMITT, FRANK W. ..... 195 Thayer St., ALR
HARVEY, N. D. ..... 112 Waterman St., OALR
LEECH, J. W. ..... 167 Angell St., OALR
MESSINGER, H. C. ..... 210 Angell St., Op.
MUNCY, WM. M. ..... 162 Angell St., OALR
PORTER, L. B. ..... 199 Thayer St., OALR
SHARP, BENJ. S. ..... 339 Thayer St., OALR
TINGLEY, LOUISA PAINE ..... 171 Westminster St., Op.
WINKLER, HERMAN A. ..... 224 Thayer St., ALR

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TOWNSEND, JOHN FERRARS ..... 85 Wentworth St., OALR

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DYERSBURG

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KNOXVILLE

KINCAID, J. H. .....421 W. Church Ave., OALR
PATTERSON, R. W. .....Medical Arts Bldg., ALR
POTTER, WILLIAM W. P. .....Medical Bldg., OALR
REAVES, ROBERT G. .....422 W. Cumberland Ave., OALR

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ANTHONY, DANIEL HARBERT .....1720 Exchange Bldg., Op.
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ELLETT, E. C. .....Exchange Bldg., Op.
EVANS, S. S. .....Physicians and Surgeons Bldg., ALR
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HARRIS, J. HARLEY .....Medical Arts Bldg., ALR
HUGHES, J. A. .....Sterick Bldg., OALR
LEVY, LOUIS .....Bank of Commerce Bldg., ALR
LEWIS, A. C. .....Exchange Bldg., OALR
LEWIS, PHILIP MERIWETHER .....Exchange Bldg., OALR
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QUALLS, HARLEY W. .....Exchange Bldg., OALR
RYCHENER, RALPH O. .....130 Madison Ave., Op.
SANDERS, SAM H. .....248 Madison Ave., ALR
SELIGSTEIN, MILTON B. ...801 Union and Planters Bank Bldg., OALR
SHEA, J. J. .....1018 Madison Ave., ALR
SIMPSON, W. LIKELY .....Phys. and Surg. Bldg., ALR
STANFORD, J. B. .....899 Madison Ave., OALR
STINSON, WM. DAVID .....805 Medical Arts Bldg., ALR
WATKINS, EDWIN DIAL .....1412 Exchange Bldg., OALR

NASHVILLE

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KENNON, W. G. ..... Doctors Bldg., OALR
ORR, EUGENE ..... Doctors Bldg., ALR
SULLIVAN, ROBT. ED. ..... Doctors Bldg., Op.
WARNER, ROBERT JAY ..... Doctors Bldg., Op.
WILKERSON, WM. W., JR. ..... Bennie-Dillon Bldg., ALR

TEXAS

AMARILLO

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